

The Effects of Altered Heat Stress on Voluntary Pacing Strategies during Prolonged Cycling

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Submitted in partial fulfillment of the requirements for the degree
Master of Science in Applied Health Sciences
(Kinesiology)

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Abstract

The Central Governor Model (CGM) suggests that perturbations in the rate of heat storage (ΔS) are centrally integrated to regulate exercise intensity in a feed-forward fashion to prevent excessive thermal strain. We directly tested the CGM by manipulating ambient temperature (T_{am}) at 20-minute intervals from 20°C to 35°C, and returning to 20°C, while cycling at a set rate of perceived exertion (RPE). The synchronicity of power output (PO) with changes in HS and T_{am} were quantified using Auto-Regressive Integrated Moving Averages analysis. PO fluctuated irregularly but was not significantly correlated to changes in thermo physiological status. Repeated measures ANOVA indicated no changes in lactate accumulation. In conclusion, real time dynamic sensation of T_{am} and integration of HS does not directly influence voluntary pacing strategies during sub-maximal cycling at a constant RPE while non-significant changes in blood lactate suggest an absence of peripheral fatigue.

Acknowledgements

First and foremost, to my supervisor Dr. Stephen Cheung, I offer my sincerest appreciation for your guidance throughout the entire process of my Master's degree. I am indebted for your knowledge and enthusiasm, while allowing me the freedom to pursue a research project independently. I attribute the calibre of my Masters Thesis to your excellence as a researcher and academic supervisor. I am truly thankful for all of the opportunities that you have provided me over the past two years.

To Dr. Andreas Flouris, thank you for all your support and advice throughout the entire process of this research project. Specifically, your help with statistical analysis and heat storage calculations was truly invaluable and without your expertise, this project would not have been a success. It was a privilege to work with such an accomplished academic.

To Dr. Mike Plyley, your wisdom and balanced approach to research has provided me with a solid foundation to my future academic career. I am sincerely grateful for the diligent and thorough feedback that you provided throughout this process, despite your many other, and likely more important commitments.

To my colleagues of the Environmental Ergonomics Laboratory: Greg McGarr, Matt Smith, Natalie Dies, Nikki Zouros, Dessi Zaharieva, and Cody Watson. Your help with the data collection was absolutely vital in the timely completion of this project; however, it is your friendship throughout all the triumphs and pitfalls of this process I will cherish the most.

Thank you to the outstanding support staff in the Faculty of Applied Health Sciences at Brock University. To Debbie Crosssthaite and Bev Minor who constantly

surpassed all that was required of you; your wealth of knowledge and constant enthusiasm truly enriched my studies at Brock University. To Greig Inglis and Raffy Dotan; your technical expertise and patience with my numerous questions and requests was greatly appreciated.

Finally, I thank my entire family: Mum, Dad, Heather, Gummie and Gramps. I am forever grateful for your unconditional love and encouragement. Through your generous financial support, I have been given the opportunity to pursue my academic goals and career aspirations. My family will always be my greatest support network and source of motivation. I hope that one day I will be able to repay you for everything that you have done for me.

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List of Abbreviations

Adenosine tri-phosphate	ATP
Ambient temperature	T_{am}
Analysis of variance	ANOVA
Auto-regressive integrated moving averages	ARIMA
Blood pressure	BP
Body surface area	A_D
Body temperature	T_{bd}
Cardiac output	\dot{Q}
Cardiovascular/Anaerobic Model	CAM
Central Governor Model	CGM
Central nervous system	CNS
Cerebral blood flow	CBF
Changes in arterial oxygen concentration	CaO_2
Coefficient of convective heat exchange	h_c
Coefficient of radiative heat exchange	h_r
Combine arm and leg exercise	cA+L
Conductive heat transfer	K
Convective heat loss transfer respiration	C_{res}
Convective heat transfer	C
Core temperature	T_c
Critical Internal Temperature Model	CIT

Electromyography	EMG
Erythropoietin	EPO
Evaporative heat transfer via respiration	E_{res}
Evaporative heat transfer via skin	E_{sk}
Heat storage	S
Heart rate	HR
Heart rate variability	HRV
Inspired oxygen content	F_iO_2
Integrated electromyography	iEMG
Lactic acid	LA
Maximal lactate steady state	MLSS
Maximal oxygen uptake	\dot{V}_{O_2}
Mean body temperature	\bar{T}_{bd}
Metabolic heat production	M
Muscle sympathetic nerve activity	MSNA
Muscle temperature	T_{mu}
Oxygen uptake	\dot{V}_{O_2}
Partial pressure of water vapor	P_{sa}
Peak oxygen uptake	\dot{V}_{O_2peak}
Peak power output	PPO
Power output	PO
Psychological Motivation Model	PMM

Radiative heat transfer	R
Rate of carbon dioxide elimination	\dot{V}_{CO_2}
Rate of heat storage	ΔS
Rate of myocardial oxygen uptake	$M\dot{V}_{O_2}$
Rate of oxygen uptake	\dot{V}_{O_2}
Ratings of perceived exertion	RPE
Rectal temperature	T_{re}
Respiration rate	RR
Skin temperature	T_{sk}
Stroke volume	SV
Sweat rate	SR
Thermal resistance	R
Ventilation expired	\dot{V}_E
Work	W

Introduction

One of the most perplexing issues in the field of exercise physiology is the mechanism(s) that contributes to a more rapid onset of fatigue during environmentally stressful exercise, such as exposure to high ambient temperatures or hypoxia. Fatigue during cycling, functionally defined as a significant decrease in power output over time (3), can be explained by several different physiological models. It is widely accepted that cardiovascular constraints limit exercise during exercise in the heat. Increased cutaneous vasculature conductance (67) and decreased plasma volume (49), combined with an increased demand in nutrient supply and waste removal from working musculature cause a competition in blood flow (98), and therefore limit exercise capacity. More recently, a model of central fatigue was proposed (103), which suggested that physiological afferents, arising from changes in ambient temperature or perturbations in the rate of heat storage, are centrally integrated in a dynamic feed-forward manner, with the end goal of regulating voluntary exercise intensity in order to maintain physiological homeostasis.

Physiological homeostasis is defined as the tendency of all systems of the human body to maintain a stable, constant condition irrespective of external stress, such as exercise or elevate ambient temperature. This state of homeostasis may be achieved in a variety of conditions, ranging from passive rest to moderate exercise. For example, physiological homeostasis may exist during exercise when the rate heat production is matched by heat loss mechanisms causing a stable core temperature, albeit elevated from baseline values (69). This state of equilibrium is achieved through the complex processing of numerous afferents and the resultant efferent feedback. The body maintains physiological homeostasis through either enhancing a present stimulus through positive

feedback or suppressing a stimulus through negative feedback. The purpose of these mechanisms is, in part, to prevent the occurrence of catastrophic collapse during stressful situations, such as exercise and environmental stress. Catastrophic collapse is defined as a substantial deviation from homeostasis, often resulting in illness, such as heat stroke or severe hypotension.

In response to ambient heat stress, this paradigm, the so-called Central Governor Model (CGM), proposes that the brain reduces muscle activation based on an increase in the rate of heat storage, with the ultimate goal of minimizing the risk of catastrophic physiological damage or collapse. While cycling in the heat, it was found that both self-paced power output and muscle activation were lower than that while cycling in normal or cool temperatures (115). Furthermore, while cycling at a constant perception of effort, it was argued that afferent feedback regarding the rate of heat storage mediated an anticipatory down-regulation in exercise intensity (113). This evidence suggests that exercise intensity is regulated in response to the rate of heat storage so that excessive heat accumulation does not occur and physiological homeostasis is maintained.

While intriguing, some existing design considerations may limit our ability to fully investigate the validity of a CGM. Chief amongst these is the need to eliminate prior knowledge of the task, as the conscious awareness of an environmental stressor being present may cause a participant to adopt a pacing strategy right from the outset of exercise based on a preconceived performance template, thereby minimizing the possibility of a sub-conscious and real-time regulation of exercise intensity. Because simply blinding subjects by presenting different environmental manipulation in a different order is ineffective in removing the knowledge of the presence of different

conditions, deception must be employed to eliminate the confounding effect of any possible psycho-physiological mechanism(s) or prior knowledge involved with this model.

The purpose of the present study was to examine the effect of a “secret” manipulation of ambient heat stress on self-paced power output during sub-maximal cycling. Using a single trial experimental design, participants cycled at a constant rating of perceived exertion (RPE) of 14 while ambient temperature was manipulated in an A-B-A pattern at 20-minute intervals between 20°C (the ‘A’ manipulation) and 35°C (the ‘B’ manipulation), and then returning to 20°C. The unique strengths of this experimental protocol are that participants were completely deceived to the true purpose of the study and blinded to the manipulation of ambient temperature. Additionally, the single trial experimental design circumvents the integrity of the repeatability of the ‘RPE clamp’ protocol, as many variables may influence power output at a given RPE from day to day. The CGM would predict that the dynamic sensation of increased ambient temperature and heat storage would cause a significant decrease in power output.

Literature Review

Models of Fatigue

One of the most perplexing issues that have faced exercise physiologists is the mechanism(s) that contributes to fatigue during whole body exercise. One of the fundamental difficulties in examining this phenomenon is the multifaceted definition of fatigue (3). From a psychological perspective, fatigue can be defined as a sensation of tiredness that contributes to decreased motivation levels in athletes (64), whereas a biomechanist would suggest that fatigue occurs subsequently with decreased muscle power output (3). Traditionally, exercise physiologists define fatigue as the failure in a specific or multiple physiological systems. The predominant model that explains fatigue from a physiological perspective is the Cardiovascular/Anaerobic Model (CAM), which proposes that fatigue occurs when the cardiopulmonary system is no longer able to supply nutrients and remove waste products from working musculature (52). However, the recent emergence of an alternative theory, the Central Governor Model, proposes that exercise intensity is regulated by a centrally located governor with the ultimate goal of preventing catastrophic physiological failure during exercise.

This controversial topic has been debated extensively within the academic community of exercise physiology; however, there is still a lack of unequivocal evidence supporting the CGM. The aim of this literature review is to summarize the theoretical framework of both the CAM and the CGM, and furthermore, to provide an unbiased interpretation of the arguments supporting and refuting each model.

The Cardiovascular/Anaerobic Model

The Cardiovascular/Anaerobic Model (CAM) proposes that fatigue occurs when the heart is no longer able to supply nutrients and remove waste products from the working musculature (3). Based on this model, physical fitness is quantified by measures of maximal oxygen uptake ($\dot{V}_{O_2 \max}$), which is the maximum amount of oxygen that an individual can transport and utilize during incremental exercise. $\dot{V}_{O_2 \max}$ values can be used to categorize individuals based on physical fitness, ranging from 30 – 50 mL kg⁻¹ min⁻¹ for untrained individuals to 70 – 80 mL kg⁻¹ min⁻¹ for elite level cyclists (75). The efficiency of the heart to transport oxygenated blood throughout the body is influenced by oxygen delivery (27, 47, 99), oxygen utilization (4, 11, 119), and metabolite accumulation (4, 15, 74).

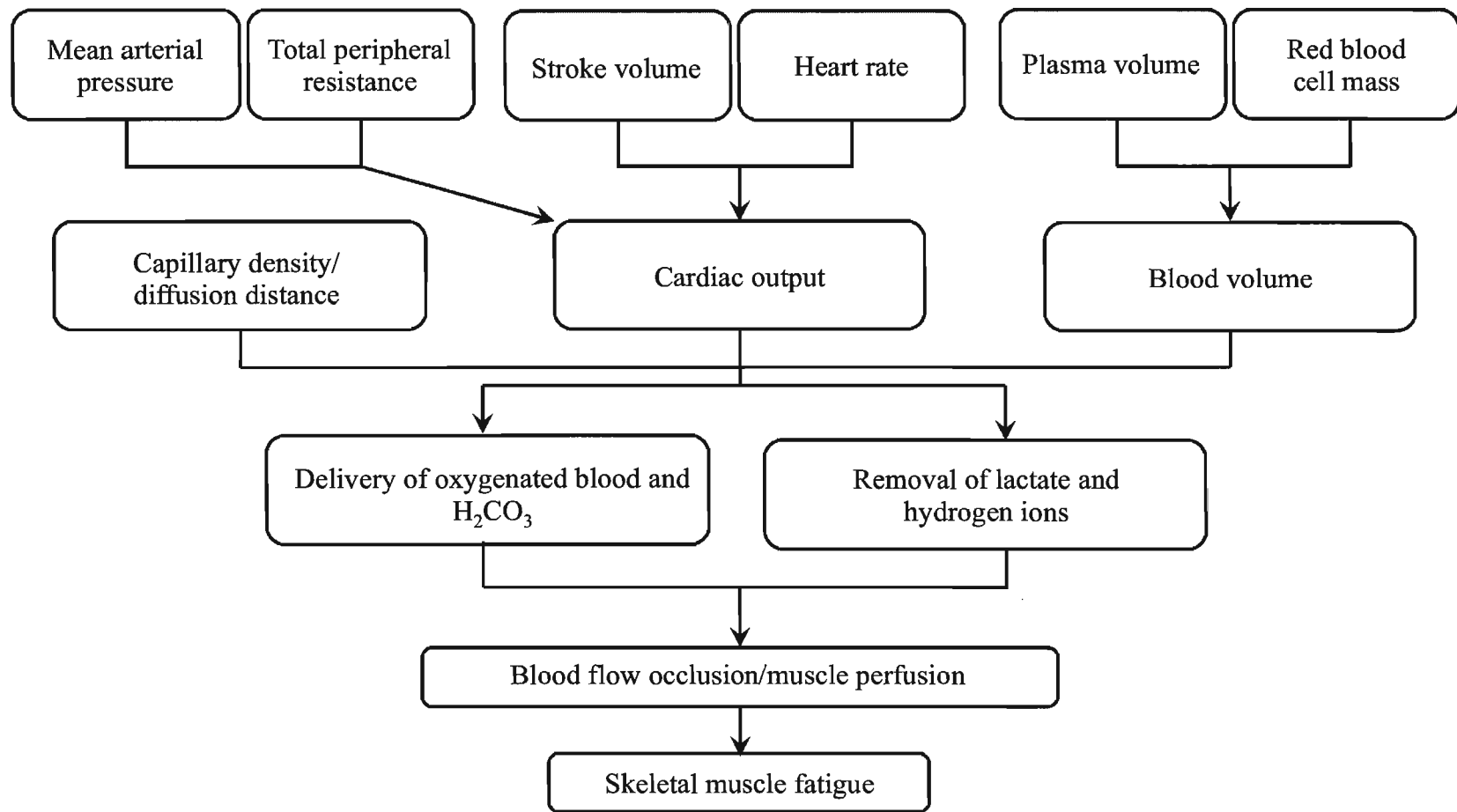
Oxygen delivery and utilization

The rate of oxygen delivery to exercising skeletal muscle is affected by several physiological variables. A significant influence of oxygen delivery is cardiac output; the volume of blood pumped by the heart each minute and is determined by the product of stroke volume and heart rate. The importance of cardiac output in the CAM was presented by Gonzalez-Alonso and Calbert (47), who examined the hemodynamic response to maximal aerobic cycling to exhaustion in a thermoneutral and heat stress environment. It was found that in each condition, both cardiac output and mean arterial blood pressure were reduced, which in turn, reduced muscle blood flow, oxygen delivery, and ultimately $\dot{V}_{O_2 \max}$.

Red blood cell mass and plasma volume have a significant effect on the ability of the cardiovascular system to deliver oxygen to the working musculature. Research has shown (27) significant differences in plasma volume content among elite athletes (6648 mL) and untrained individuals (4876 mL); however, artificial increases in blood plasma in elite cyclists has been shown to have no effect on $\dot{V}_{O_2 \max}$ (121). Upon further investigation, it was found that plasma volume expansion increased both cardiac output and stroke volume. This may imply that exercise at $\dot{V}_{O_2 \max}$ is limited by blood volume and haemoglobin content, and not necessarily by the capacity of the heart to circulate blood (121).

The importance of red blood cell mass to oxygen delivery is supported by evidence of increased physical capacity following red blood cell reinfusion (blood doping) or exogenous erythropoietin (EPO) supplementation (16). EPO is a naturally occurring hormone that is secreted from the kidneys and is accentuated in response to hypoxia localized to the arteries. Like blood doping, these practices artificially increase the haemoglobin content of the blood, and therefore, the oxygen carrying capacity of the blood (21). Hematocrit, the fraction of total blood volume that is occupied by red blood cells, when excessively high can be lethal as it increases the viscosity of the blood. This can cause cardiovascular failure, such as stroke, heart attack, or pulmonary edema (10).

Figure 1. Schematic representation of the Cardiovascular/Anaerobic Model. Delivery of nutrients and removal of waste products is affected by capillary density, cardiac output and blood volume that results in skeletal muscle fatigue (3).



The third factor that will influence oxygen delivery to working muscles is blood flow occlusion. Research indicates that during sub-maximal cycling, there is a linear relationship between muscle blood flow and power output (99). This conclusion is logical as the delivery of nutrients to the muscle is essential for physical performance. However, during tetanic muscle contractions, the occlusion of blood flow is much greater, and therefore will have a negative effect on physical performance. Takaishi et al. (107) indicate that when intramuscular pressure is greater than the local blood pressure, muscle blood flow becomes compromised during exercise.

The control of blood flow through skeletal muscle is modulated by chemical, neurological and mechanical factors, accounting for a 20-fold increase observed during exercise (99). The initiation of exercise causes an immediate decrease in regional oxygen content due to the metabolic demands of the working muscle mass. In response, arterial vasodilatation occurs to maintain the contractile integrity of the local vasculature, thus increasing blood flow. Other vasodilators, such as potassium, ATP, lactic acid, and carbon dioxide are believed to influence muscle blood flow; however, the role of each substance remains unknown. The autonomic nervous system regulates muscle blood flow via sympathetic vasoconstrictor nerves, specifically by secreting norepinephrine to skeletal muscle. Norepinephrine acts directly on inactive musculature, causing vasoconstriction in local blood vessels and therefore, redistributing blood flow to active muscles (99).

There are several circulatory readjustments occurring during exercise that influence the rate of oxygen delivery to skeletal muscle. Initially, the onset of exercise causes a sympathetic discharge, which results in an increase in heart rate, vasodilatation

of active muscle and vasoconstriction of inactive muscle. The subsequent increase in pressure caused by the narrowing of the veins contributes an increase in the mean systemic filling pressure, promoting the return of blood to the heart. Ultimately, these mechanisms contribute to an increase in arterial pressure during exercise and therefore, an increase in cardiac output.

A strong correlation between endurance training and the oxidative potential of skeletal muscle has been well documented in recent studies. Skeletal muscle experiences significant adaptations in response to endurance training, resulting in larger and more abundant mitochondria (119), increased enzyme activity (11), greater capillarisation (95), and increased myoglobin content (92). The physiological adaptations that occur as a result of exercise increase the capacity of the mitochondria to produce adenosine triphosphate (ATP), the main transporter of chemical energy during metabolism (11, 119).

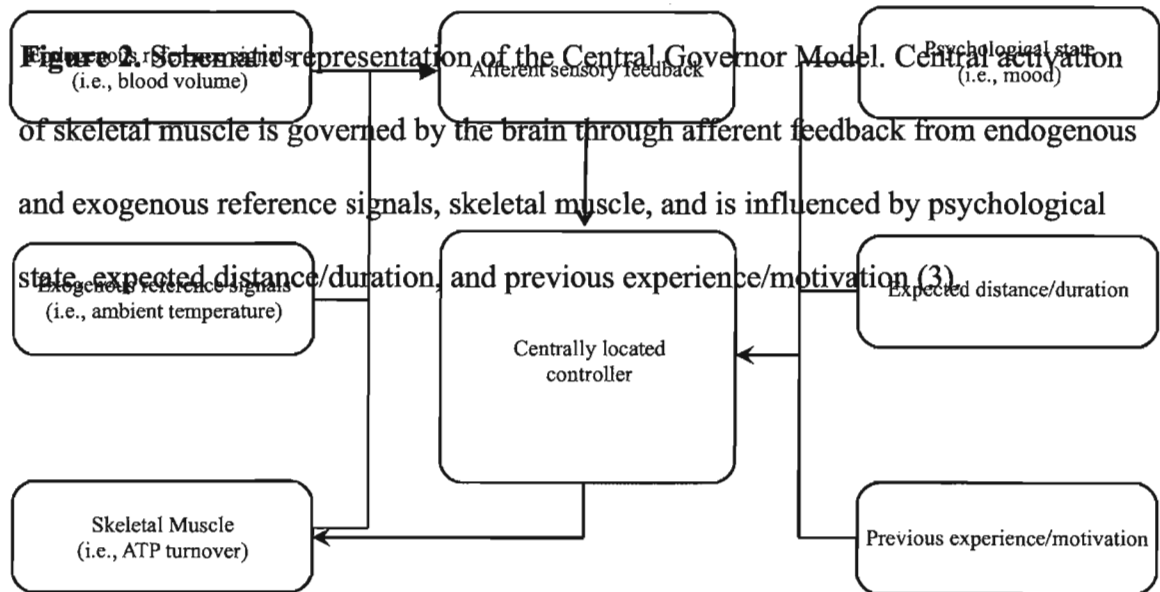
Metabolite Accumulation

The third variable that will influence the ability of the cardiovascular system to remove waste products from working musculature is the metabolite accumulation that occurs during exercise. The primary waste product produced during high intensity exercise is lactic acid. The accumulation of this metabolite occurs when its rate of production exceeds its rate of removal, allowing for the dissociation of lactic acid into lactate and hydrogen ions. The resultant decrease in pH has a detrimental effect on muscle contraction as it inhibits calcium release during the cross bridge cycle (15). Many studies indicate a negative relationship between lactate accumulation and power output during cycling (4, 74).

The Central Governor Model

The Central Governor Model (CGM) proposes that the central nervous system continuously integrates afferent feedback to regulate exercise intensity, ultimately aiming to safely and efficiently complete a task without reaching a state of catastrophic physiological failure (103). These feedback signals contain kinetic and metabolic information from the body and, when integrated with central senses, regulates an appropriate amount of neural drive to the exercising muscle mass via afferent pathways (116). It is suggested that this model of fatigue acts as an anticipatory mechanism to protect vital organs from catastrophic failure during exercise (85).

One of the fundamental principles of the CGM is the theory of telo-anticipation, as originally proposed by Ulmer (116). Telo-anticipation is the sub-conscious calculation of an appropriate pacing strategy that elicits the highest possible power output while maintaining physiological homeostasis throughout the entire period of physical activity. This complex calculation accounts for the metabolic requirements of the given task, taking into account the ambient environmental conditions and current physical and psychological status of the body. The sensation of fatigue would be the conscious presentation of altered muscle activation, and therefore, should be considered as dynamic, opposed to an absolute event during exercise (103).



Origins of the Central Governor Model

The original model of central fatigue was first proposed by Hill et al. (57, 58). They found that exercise was limited by cardiac and cerebral distress, as opposed to physiological limitations in skeletal muscle. It was believed that, as a result of intense exercise, increases in blood hydrogen-ion concentration and decreases in blood oxygen saturation caused a decrease in cardiac output. Hill et al. argued that as excessive exertion of the heart is wasteful, this complex mechanism aimed to maintain appropriate oxygen saturation levels of the blood.

Hill et al. (58) were the first to test this model using alterations in inspired oxygen content ($F_{I}O_2$) to observe the effects on exercise performance. The results of these experiments indicate that the inhalation of hyperoxic air allows for much higher oxygen intake and exercise performance. However, Hill et al. (58) hypothesized that this substantial increase in performance cannot be adequately explained by an increase in oxygen saturation alone, but by a governor that acts in accordance with the level of blood oxygen saturation.

The original model of central fatigue, as proposed by Hill et al. (57, 58), has received little attention until its re-emphasis by Noakes (84, 85). Noakes argues that the traditional CAM of fatigue fails to address four critical events during exercise, and that CGM satisfies these observations. According to Noakes, the shortcomings of the CAM of fatigue are that:

- i. it is far more likely that the heart will experience anaerobiosis before skeletal muscle;
- ii. there is no scientific evidence for anaerobiosis, hypoxia, or ischemia in skeletal muscle during maximal exercise;
- iii. there is no explanation for the termination of exercise in conditions, such as prolonged exercise, hot and hypoxic environments, and disease, without evidence of anaerobiosis, ischemia, hypoxia, and full neuromuscular activation of skeletal muscle; and
- iv. cardiovascular measures of fitness, such as $\dot{V}_{O_2 \max}$ and anaerobic threshold, are indifferent among athletes of similar abilities.

Regulation of exercise intensity: Central or peripheral?

Although the Cardiovascular/Anaerobic Model is widely believed to be the contemporary view of fatigue during physical activity, the emergence of the Central Governor Model has questioned some of the fundamental arguments supporting this theory. The primary argument supporting the Central Governor Model is that physiological homeostasis will be maintained at volitional fatigue, and this is further supported by changes in skeletal muscle activation associated with altered environmental conditions (111, 113, 115). Although these observations support an anticipatory reduction in muscle activation, many believe that this model cannot be substantiated by various observations of the \dot{V}_{O_2} and neuromuscular responses to maximal exercise, and the blood perfusion response to exercise.

The Central Governor Model has been a highly debated topic in the scientific community; however, several academic studies support an anticipatory reduction in central drive, as proposed by this novel theory. Noakes and colleagues at the University of Cape Town have conducted the majority of the research supporting this controversial theory. Very little support for this model has been shown outside this academic circle. Many experts claim that the reported evidence for the Central Governor Model is plagued by flawed methodologies and interpretation, such as heat storage calculations based on thermometric measurements and the reliance on the 'RPE clamp' protocol (34); however, it is extremely difficult to test voluntary central control due to the potential psychological nature of this theory.

Physiological Homeostasis at Volitional Fatigue

A fundamental argument supporting the CGM is the lack of a catastrophic physiological failure at volitional fatigue during maximal exercise (90). During exercise, feedback from vital organs and working musculature is delivered via afferent feedback loops to the central nervous system, and as a result, muscle activation is appropriately altered. This anticipatory change in muscle activation protects the body's physiological systems from cardiovascular, metabolic, respiratory, and thermoregulatory distress, and ultimately catastrophic collapse.

Baron et al. (9) support the theory of a physiological reserve. In a study examining cycling to exhaustion at an intensity corresponding to maximal lactate steady state (MLSS), Baron et al. (9) reported that measurements of oxygen uptake ($\dot{V}_{O_2 \max}$), carbon dioxide output (\dot{V}_{CO_2}), respiratory rate (RR), and minute ventilation (\dot{V}_E) obtained upon completion of the test to exhaustion at MLSS were significantly lower than values obtained during an incremental exercise test. These findings support the existence of a physiological reserve capacity, and therefore, cannot be considered as factors contributing to fatigue during exercise at MLSS. A significant increase in heart rate was observed throughout the duration of the trial. The authors (9) hypothesized that this increase may be influenced by increases in sympathetic nervous activity, increases in circulating norepinephrine, and elevations in core body temperature. These observations parallel assumptions made by the Central Governor Model, as the stress of exercise may induce a form of fight-or-flight response in the sympathetic nervous system in an attempt to regulate exercise intensity. Furthermore, cardiac drift is a common occurrence during

exercise in the heat, which causes a gradual increase in heart rate over time. These factors can contribute to the increase in heart rate observed throughout the duration of the trial.

In order to eliminate the confounding effects of heat storage during exercise to exhaustion at MLSS, a modification in an experimental protocol may produce more appropriate results. Research conducted by Galloway and Maughan (43) indicates that, in stationary cycling with minimal air movement, ambient temperatures of 11°C are optimal to maximize exercise tolerance time. Although core temperatures upon completion of exercise ($38.4 \pm 0.7^{\circ}\text{C}$) are significantly lower than what has been assumed to be a critical limiting body temperature ($>40^{\circ}\text{C}$) (23), the rate of change in heat storage may contribute to fatigue and should be considered as a confounding variable.

A significant limitation of the study conducted by Baron et al. (9) is the lack of statistical support for the conclusions drawn in the study. The crux of the argument made in this paper is that the physiological variables measured during MLSS testing are significantly lower than those obtained during incremental testing, an indication of the physiological capacity (9); however, there is a lack of statistical support for this conclusion. The authors (9) indicate that a repeated measures analysis of variance (ANOVA) suggests a significant difference between the two conditions ($p < 0.05$), but the focus of the paper was primarily on the changes in each individual variable during the MLSS test. Adequate support for the main conclusion, that there is a significant difference in physiological measures at volitional fatigue and physical capacity, is essential to properly supplement the conclusions drawn in this paper.

The notion of centrally regulated exercise capacity and the influence of perception of effort are supported by improved athletic performance when ingesting acetaminophen

(ACT) prior to a 16.1 km time-trial. Mauger et al. (77) established that, when compared to a control group, the ingestion of ACT caused an increase in mean power output, specifically during the middle portion of the time trial, resulting in an increase in exercise performance. Furthermore, it was noted that there was an increase in heart rate and blood lactate production during the ACT trial; however, a significant increase in the rating of perceived exertion and pain did not occur. These observations suggest that the inhibition of pain sensitivity allows athletes to work at an intensity closer to their physiological threshold, thereby over-riding the central mechanisms that regulate exercise (77). These results support the existence of a central governor, given the up-regulation in exercise performance and subsequent increase in peripheral stress (increased heart rate and blood lactate production) without any increase in perception of effort or pain.

A strong association between the perception of fatigue and neurotransmitter activity and sensitivity in the brain highlights the importance of central fatigue during exercise. Specifically, serotonin (5-HT) and dopamine (DA) has been shown to fluctuate during exercise and ambient heat stress, causing changes in arousal and initiating volitional fatigue (80). Pharmaceutical regulation of serotonin using an animal model is known to influence endurance capacity; supplementation of a 5-HT agonist has shown to decrease endurance capacity, while a 5-HT antagonist has been shown to increase endurance capacity (29). Additionally, the arterial concentration of DA is elevated during exercise and hyperthermia, while a sharp decrease has been associated with volitional fatigue during exercise. This evidence supports the paradigm of central fatigue as exercise capacity appears to be regulated by mechanisms originating inside the CNS as opposed to peripheral factors.

Presence of an end-spurt

Possibly the most convincing evidence supporting the CGM is the conscious up-regulation of exercise intensity when in close proximity to task completion. This phenomenon, known as an end-spurt, is often displayed during long distance running events (112) irrespective of environmental conditions (115). Given the unpredictable nature of athletic competition and therefore the varying physiological demands to complete the required task, athletes pace themselves so that they maintain a reserve capacity. As the athlete nears completion of the event, the degree of uncertainty diminishes, therefore allowing a safe increase in exercise intensity (114).

Performance improvements when in close proximity to task completion have been observed during a multitude of tasks. Catalano (18) observed a significant improvement in performance during vigilance and rotary pursuit tracking tasks when participants were told that they were in close proximity to task completion. Participants were separated into two groups; the experimental group was shown a red light when they had completed 90% of the task, while the control group was shown a red light at random times during the trial. The experimental group displayed a significant improvement in performance beyond the 90% point of task completion, whereas the control group did not show any improvement (18).

The mechanism(s) underlying the end-spurt phenomenon appears to be associated with increases in arousal and motivation (18). When an athlete senses or is informed that he or she is in close proximity to completing the event, an increase in vigour causes an increase in exercise performance despite the environmental conditions or physiological state (115). Consequently, this up-regulation in exercise intensity is caused by a change in

psychological status and may occur irrespective of the physiological afferents being relayed to a centrally located controller.

An alternative explanation of the end-spurt phenomenon is proposed by the Psychological/Motivational Model (PMM) (109). This theory is similar to that of Noakes (85, 86, 90), except that, instead of a subconscious reduction in muscle activation, the PMM proposes that pacing strategies are altered intentionally (3). Catalano et al. (18-20) have examined the end-spurt in performance in simple motor tasks, and have found that the perceived proximity to task completion causes higher motivation, and therefore, the end-spurt in performance. Although the results of these experiments (18-20) provide evidence supporting an increase in pace during the final 10% of a motor task, the validity of these results must be considered when applied to whole body exercise.

The presence of an end-spurt remains highly debated, as conflicting results have emerged in recent studies. In opposition to Noakes' views (86), Ely et al. (35) indicate that highly successful marathoners maintain a more consistent pace throughout the race, whereas novice athletes are more likely to vary their pacing selection. It was found that those runners who were able to maintain a consistent pace were less likely to produce an end-spurt.

Other models of fatigue (13, 14) suggest that pacing strategies are dictated by the physiological capacity of the body, and therefore cannot explain the phenomenon of the 'end-spurt'. The challenge when critically analyzing different models of fatigue is to establish a direct comparison that can cleanly separate and isolate the predictions of each theory. Although there is no evidence proposed by the Cardiovascular/Anaerobic Model that supports the presence of an end-spurt during maximal exercise, the actual intensity at

which a marathon is run must be considered. Most marathon runners choose a pace that is far below maximal intensities ($65 - 85\% \dot{V}_{O_{2\max}}$), and therefore, the source of peripheral fatigue is glycogenolysis, not anaerobic metabolism (26). Given that this exercise intensity is not maximal by any definition, it is plausible that an end-spurt can be explained by a shift from glycogenolysis to anaerobic metabolism, therefore providing the necessary energy for an end-spurt.

When examining pacing selection during a marathon, further evidence supports the presence of a physiological reserve when an athlete completes a race. Noakes (86) notes that upon completion of a marathon, highly exhausted athletes still have the capacity to walk, and therefore, could not have exercised to their true physiological capacity. In addition to the presence of an 'end-spurt', this phenomenon cannot be explained by any other model of fatigue than the Central Governor Model. This aspect of the Central Governor Model is often questioned as a small number of marathon finishers do exhibit catastrophic failure upon completion of the event. Kenefick et al. (66) report that 0.2 - 3.7% of participants collapse during distance running events, due to a variety of potential issues, such as physical exhaustion, hyperthermia, dehydration, hypothermia, and postural hypotension. Although proponents of the CGM would argue that individuals who experience such physiological catastrophe during an athletic event suffer from a pre-existing disease/injury, this evidence appears to be a point of contention for those who support the CAM. Presently, there is no conclusive evidence implicating disease/illness in unsuccessful marathoners, and therefore cannot support the Central Governor Model when considering these issues.

Muscle activation and hyperthermia

A fundamental facet of the CGM is that environmental stressors, such as altered inspired oxygen content (111), and a change in the rate of heat storage (113, 115), modulate muscular recruitment during exercise. Tucker et al. (113, 115) have examined the effect of hyperthermia and the resultant rate of heat storage on exercise performance in response to several thermal environments. The result of their research program indicates that there is a decreased exercise capacity associated with hyperthermia (113), and that an anticipatory reduction in muscle activation may coincide with an increased rate of heat storage (115).

Anecdotal and scientific evidence (43) indicate that exercise capacity and performance is decreased in the heat; however, the mechanism(s) that cause this phenomenon are unclear. Tucker et al. (115) aimed to test the hypothesis that muscle activation is altered in response to either hot (35°C) or cold (15°C) environments during dynamic exercise. Experienced cyclists were recruited and performed a 20 km self-paced time trial in both hot (HOT) and cold (COOL) conditions, and only received feedback regarding total distance covered at 1 km intervals throughout exercise.

The results of the Tucker et al. study (115) clearly indicate a decreased exercise capacity during the hot environment, as both exercise time and power output were impaired in this stressful environment (28.8 ± 1.8 min in COOL vs. 29.6 ± 1.9 min in HOT, $p < 0.001$; 272 ± 45 W in COOL vs. 255 ± 47 W in HOT). Furthermore, it was found that power output decreased progressively throughout HOT, whereas power output was maintained throughout the duration of the COOL trial. Integrated electromyography (iEMG) data indicate a higher muscle activation ($p < 0.05$) in COOL compared to HOT at

both 10 km and 20 km into the required distance. The thermoregulatory response was found to be insignificant as both rectal temperature, and the rate of heat storage was similar in both trials.

One of the most significant findings of this study (115) is that homeostasis was never compromised as muscle blood flow and metabolism are not limited during exercise. Additionally, results indicate that muscle activation is altered, and it is believed that the objective of this mechanism is to ensure that the rate of heat storage is similar in both the HOT and COOL conditions. Although the stress induced by the HOT environmental condition was higher than that of the COOL condition (15°C vs. 35°C), rectal temperatures were not significantly different between each trial. Tucker et al. (115) proposed that the evidence provided by this study indicates that a centrally located governor regulates exercise intensity through altered muscle activation to ensure that excessive heat storage does not occur in hot environmental conditions.

Tucker et al. (113) conducted a follow up study to support their previous evidence of a central governor that regulates muscle activation in the heat. Using a unique experimental protocol termed the “RPE Clamp”, participants were instructed to cycle at a set rating of perceived exertion (RPE) for as long as possible until power output fell below acceptable levels. Participants were instructed to vary their work intensity so that they maintain a constant RPE throughout the entire duration of the trial. Each participant was subjected to three environmental conditions: COOL (15°C), NORM (25°C), and HOT (35°C).

The results of the study (113) indicate that while the power output decreased in a linear fashion, the rate of decline was largest in the HOT condition compared to NORM

and COLD ($2.35 \pm 0.73 \text{ W} \cdot \text{min}^{-1}$, $1.63 \pm 0.70 \text{ W} \cdot \text{min}^{-1}$, $1.62 \pm 0.80 \text{ W} \cdot \text{min}^{-1}$, respectively). Additionally, the total duration of exercise was significantly lower ($p < 0.05$) in the HOT condition, compared to both COOL and NORM. The rate of heat storage was only significantly higher in the HOT condition, although after 4 minutes no difference in heat storage was found between conditions. Rectal temperature was found to be significantly higher in the HOT condition during the last 20% of exercise compared to NORM and COOL.

In conclusion, Tucker et al. (113) indicate that fatigue occurs as a result of hyperthermia, and modifications in central muscle activation caused a protective decrease in exercise intensity. It was found that the reduction in muscle activation was influenced primarily by the rate of heat storage, as opposed to a higher core body temperature. Furthermore, the timing at which the reduction of muscle activation occurred was shortly after the onset of exercise, suggesting that this mechanism acts in anticipation of any peripheral physiological changes. Tucker et al. (113) concluded that this evidence supports the existence of a central governor as exercise intensity during the heat is influenced by muscle activation, rather than peripheral factors.

Although the evidence provided by Tucker et al. (113, 115) provides seemingly convincing evidence supporting the CGM, many experts in the field postulate that the interpretation of these results are fundamentally flawed (62). An alternative explanation for the increased rate of heat storage upon the onset of exercise is proposed by Jay and Kenny (62), who suggest that energy release due to contracting musculature is immediate and that the compensatory mechanisms are significantly delayed. This information

provides an alternative explanation for the results obtained by Tucker et al. (113), although it provides no support for the CGM model.

Furthermore, many experts question the methods used by Tucker et al. (113, 115), and suggest that several fundamental flaws may plague the conclusions obtained in the studies. Although some publications claim that the “RPE Clamp” (110) is a reliable technique, others have yet to reproduce this technique consistently in other studies. Also, many thermal physiologists indicate that thermometric calculations, as employed by Tucker et al. (113, 115), are inadequate for this application, and suggest that calorimetry would be a more appropriate technique for measuring the rate of energy expenditure from the human body to the environment (or vice versa) (62).

In agreement with evidence provided by Tucker et al. (113, 115), physiological and perceptual responses to self-paced 100-km cycling suggests that exercise intensity is down-regulated in response to heat stress (1). The main finding of the study was that muscle activation and power output declined in the heat prior to any significant increase in rectal temperature, thereby supporting the concept of an anticipatory reduction in exercise intensity. More interesting, however, is the correlation between the increase in thermal sensation and the resultant decline in power output, and the lack of a relationship between power output, ratings of perceived exertion and perceived pain (1). This evidence further implicates the perceptual influence of ambient heat stress on self-selected pacing strategies, and suggests that perception of exertion and pain are not suitable analogues of thermal stress during exercise.

$\dot{V}_{O_2 \max}$ *response to maximal exercise*

One of the most significant questions that perplex exercise physiologists is the mechanism(s) that limit $\dot{V}_{O_2 \max}$ during whole body exhaustive exercise. According to the CAM, $\dot{V}_{O_2 \max}$ is limited by the capacity of the cardiovascular system to circulate blood throughout the body during maximal exercise. The value of $\dot{V}_{O_2 \max}$ can be quantified as the product of cardiac output and arterial oxygen content (32, 33). Given this definition, physiological variables, such as haemoglobin, F_iO_2 , and mode of exercise, can modify the measured values of $\dot{V}_{O_2 \max}$, but do not determine the upper limit of $\dot{V}_{O_2 \max}$ during whole body exercise (14).

To provide evidence supporting a myocardial limit of $\dot{V}_{O_2 \max}$ during exercise, Brink-Elfegoun et al. (14) examined the response of blood pressure (BP), cardiac output (\dot{Q}), and heart rate (HR) during various rates of maximal work. Previous research (71, 82) indicates that BP multiplied by \dot{Q} and BP multiplied by HR can be used to approximate a measure of the oxygen uptake of the myocardium ($M\dot{V}_{O_2}$). The authors hypothesize that while exercising at different maximal intensities, BP would increase accordingly with effort, while HR, \dot{Q} , and $\dot{V}_{O_2 \max}$ would remain unchanged throughout each test (14).

All participants performed a combined arm and leg (cA+L) exercise (14), as work isolated to either the upper or lower body was not stressful enough to elicit a true maximal \dot{V}_{O_2} value (34). Given the assumption that all participants will achieve a plateau in \dot{V}_{O_2} , extensive pre-testing was conducted to determine the lowest work rate that

corresponds to a $\dot{V}_{O_2 \max}$ value. This value was then used as a reference for the experimental protocol during which the low maximal exercise test (L) was conducted at this intensity, with the high maximal exercise test (H) being conducted at a work rate ~10% higher than that of L (14).

The results of the study (14) indicate that $M\dot{V}_{O_2}$ was higher in H trials compared to L, due to increases in both systolic blood pressure and calculated mean blood pressure. This finding suggests that the mechanical work of the heart is increased during the higher intensity maximal exercise, as changes in $\dot{V}_{O_2 \max}$, heart rate or cardiac output were not observed. Furthermore, it is speculated that the difference in exercise time between the two maximal exercise trials is caused by an increase in the oxygen demand of the heart, ultimately reducing the amount of oxygen available for the working skeletal muscle (14). The authors state that these results refute the Central Governor Model, in that the individuals were capable of working at intensities higher than $\dot{V}_{O_2 \max}$, and further that $\dot{V}_{O_2 \max}$ is determined by the capacity of the heart to transport oxygenated blood to the periphery during whole body exercise (14).

Further evidence provided by Stray-Gundersen et al. (105) supports the Cardiovascular/Anaerobic Model of fatigue, proposing that $\dot{V}_{O_2 \max}$ is determined by the capacity of the heart to circulate oxygenated blood to the periphery. The authors examined the effect of a pericardiectomy procedure on untrained dogs subjected to sub-maximal and maximal exercise, while measuring \dot{Q} and \dot{V}_{O_2} . It was hypothesized that the pericardium reduces the compliance of the myocardium; therefore, removing the

pericardium may increase end-diastolic blood pressure, and ultimately $\dot{V}_{O_2 \max}$. In conclusion, it was found that by removing the pericardium, $\dot{V}_{O_2 \max}$ and cardiac output were increased significantly during maximal exercise. This study (105) supports the Cardiovascular/Anaerobic Model by reinforcing the hypothesis that $\dot{V}_{O_2 \max}$ is limited by the capacity of the heart to circulate blood to the periphery.

When examining the response of \dot{V}_{O_2} to maximal exercise, Brink-Elfegoun et al. (14) indicate that stroke volume reaches a plateau at a sub-maximal intensity. Although the response during exercise remains equivocal, recent research indicates that, although not linear, stroke volume increases progressively throughout incremental exercise in numerous populations (118). Given that the participants recruited for the study were young (24.4 ± 2.8 years) and highly fit individuals ($59.1 \pm 2.0 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$), recent research (46, 56) would suggest that stroke volume would not reach a plateau through this exercise protocol. Given the lack of conclusive evidence, it is plausible that cardiac output, a function of stroke volume, may differ when comparing different work rates at $\dot{V}_{O_2 \max}$, given different populations or testing protocols.

In a rebuttal (89) to the conclusions made by Brink-Elfegoun et al. (14), Noakes and Marino indicate that a higher blood pressure at the same \dot{Q} suggests that a blood pressure raising mechanism occurs during exercise at 120% of $\dot{V}_{O_2 \max}$; the higher afterload that occurs during 120% $\dot{V}_{O_2 \max}$ indicates that the heart is not working maximally at 100% $\dot{V}_{O_2 \max}$. Furthermore, Noakes and Marino indicate that the amount of work standardized for time is greater during exercise at 100% $\dot{V}_{O_2 \max}$, although the heart

works harder during 120% $\dot{V}_{O_2 \max}$. It is suggested that this evidence “dissociates the amounts of work performed by the heart and the skeletal muscle” (89) and disproves the Cardiovascular/Anaerobic Model.

Neuromuscular response to maximal exercise

Further evidence supporting the Cardiovascular/Anaerobic Model is provided by a recent study conducted by Brink-Elfegoun et al. (13) examining the neuromuscular and circulatory adaptations during maximal intensity exercise. The purpose of the study was to refute the Central Governor Model (73, 91, 103) and was based on the belief that oxygen uptake can reach a plateau during maximal exercise and that individuals can continue to work following the achievement of this plateau. Using the combined arm and leg (cA+L) model, the electromyographical (EMG) response to maximal exercise was examined at three different work rates. The results of this study (13) indicated that all participants reached a \dot{V}_{O_2} plateau during whole body exercise, evidence that refutes the Central Governor Model. Furthermore, $\dot{V}_{O_2 \max}$ values obtained in all maximal work exercises were similar despite significant differences in work rate and EMG activity increases during the \dot{V}_{O_2} plateau.

Analysis of $\dot{V}_{O_2 \text{peak}}$ values obtained in separate arm and leg exercise protocols provide further evidence that indicate that the heart is the limiting factor during whole body exercise. Initial pre-testing indicates that $\dot{V}_{O_2 \text{peak}}$ values are $3.32 \text{ L} \cdot \text{min}^{-1}$ and $4.46 \text{ L} \cdot \text{min}^{-1}$ for incremental arm exercise and incremental leg exercise, respectively. The summation of these two values represents the theoretical $\dot{V}_{O_2 \max}$ value during whole body

exercise; however, this value ($7.78 \text{ L} \cdot \text{min}^{-1}$) is much greater than the $\dot{V}_{O_2 \text{ max}}$ value obtained during cA+L exercise. This 40% discrepancy between the theoretical and actual $\dot{V}_{O_2 \text{ max}}$ value obtained during maximal cA+L work indicates that the limiting factor during exercise is not peripheral oxygen utilization, but the capacity of the heart to circulate blood.

Although research conducted by Brink-Elfegoun et al. (13) suggests that exercise capacity during whole body exercise is limited by central circulatory factors (rather than peripheral factors), this evidence does not completely refute the Central Governor Theory. Noakes and Marino (88) indicate that although the heart may limit $\dot{V}_{O_2 \text{ max}}$ during whole body exercises, ultimately $\dot{V}_{O_2 \text{ max}}$ or the capacity of the heart to circulate blood to the periphery is regulated by the number of motor units recruited during exercise (89). Thus, a central governor acts in anticipation by reducing muscle activation to prevent cardiac ischemia during maximal exercise. Brink-Elfegoun et al. (13, 14) do not provide adequate evidence to directly refute this theory.

Blood perfusion during exercise

One of the most significant factors that dictates exercise capacity during maximal intensity exercise is oxygen delivery, as a function of muscle blood flow occlusion. During sub-maximal exercise, the cardiovascular system maintains a homeostasis between oxygen supply and oxygen demand in working musculature (50); however, this equilibrium may be compromised during whole body maximal exercise (81). To examine any possible occlusion of blood flow during whole body exercise, Mortensen et al. (81) examined systemic and peripheral hemodynamic and cardiovascular functions during

supramaximal exercise. The results of the study indicate that muscle perfusion, vascular conductance, oxygen delivery, and aerobic metabolism are limited during high-intensity exercise, which parallels a plateau in cardiac output. Furthermore, it was found that there was an upper limit to muscle perfusion irrespective of the energy demands of the muscle tissue. This would suggest that both the cardiovascular system and peripheral vasoconstriction limit the metabolic demand of working skeletal muscle during maximal exercise (81).

Mortensen et al. (81) postulate several explanations for the decrease in muscle blood flow that occurs during maximal and supramaximal exercise. It is hypothesized that both central and local factors may limit blood flow to skeletal muscle due to feedback from metabolic, thermal, mechanical, and vascular events. Additionally, increased local vasoconstrictor and decreased vasodilator activity may contribute to diminished muscle perfusion. Possible mechanisms include increases in muscle sympathetic nerve activity (MSNA), and circulating noradrenalin during exercise, and increase sympathetic vasoconstrictor activity during exercise (81).

Although the authors indicate that these results refute the Central Governor Model, a different interpretation of the findings may indicate otherwise. As mentioned above, the authors indicate that a plateau in blood perfusion “*might be the result of the interaction of local and central reflexes signalling metabolic, thermal, mechanical, and vascular events in many regions of the body including skeletal muscle, heart and brain*” (81). This statement could be reinterpreted to support the Central Governor Model, as it is suggested that “central reflexes” regulate the cardiovascular system. Furthermore, one issue that was not addressed in this publication was the influence of intra-musculature

pressure that occurs during cycling. Takaishi et al. (108) indicate that the force created by muscular contractions during cycling increases the pressure within the muscle, therefore limiting blood flow through the tissue.

Hyperthermia and Exercise

It is well recognized that heat exposure has a detrimental effect on many aspects of the human body, causing such issues as hypohydration, changes in metabolic rate, cardiovascular dysfunction, thermoregulatory collapse, and a decreased exercise capacity (43). More recent research postulates that a decreased exercise capacity occurs as a result of central factors, such as voluntary exhaustion which occurs at a critical core temperature, irrespective of peripheral factors (83). Therefore, the detrimental effect of hyperthermia on exercise capacity can be discussed in terms of peripheral and/or central limitations.

Peripheral limitations

The distribution of blood throughout the human body is necessary for normal functioning of cells. When humans are subjected to high ambient temperatures, vasodilatation of the peripheral vasculature occurs as a mechanism to dissipate heat (22); however, when individuals are active, there is also a substantial demand for blood supply to the active musculature to meet the energy demands of exercise. When humans engage in exercise in the heat, the overwhelming demand for blood supply by these two physiological systems ultimately result in a decreased exercise capacity (43), thermoregulatory failure (22), or both.

The blood flow capacity of working muscle during exercise is enormous. It is estimated that during maximal intensity running, which utilizes approximately 15 kg of

muscle mass, would require a cardiac output of $40 \text{ L} \cdot \text{min}^{-1}$ to fulfil the required demand of muscle vasodilatation (48). This demand for cardiac output, which only represents the demand of the working musculature, far exceeds the capacity of an individual of average physical capacity. Furthermore, blood flow requirements of the skin during a thermally stressful environment can exceed rates of $8 \text{ L} \cdot \text{min}^{-1}$.

From a cellular perspective, exercise in a hot environment can have a detrimental effect on blood composition, specifically by reducing blood plasma volume through sweating. Plasma volume, combined with the quantity of red blood cell mass, directly influences the total blood volume and ultimately the delivery of nutrients and removal of waste products. A reduction in blood volume caused by hypo-hydration would therefore decrease exercise capacity in the heat.

Central limitations

Nielsen et al. (83) examined the effect of heat acclimatization on exercise tolerance in the heat. Although not the primary focus of the study, it was observed that all participants reached volitional fatigue at a consistent internal temperature of 39.7°C , irrespective of their heat acclimation status. Furthermore, no decreases in cardiac output, blood flow, or metabolite accumulation were observed. These observations caused a progressive paradigm shift in our thinking about human thermoregulation, leading to the development of the Critical Internal Temperature (CIT) Model, in which higher core temperatures were believed to be the cause of exhaustion during exercise in the heat (22).

Cheung and McLellan (24, 25) supported the CIT model; they observed that volitional fatigue occurs in moderately fit individuals at a consistent terminal core temperature, regardless of hydration and acclimation status. A novel finding of this study

(25) was that higher levels of aerobic fitness increased the critical internal temperature (T_{crit}), therefore increasing the exercise capacity of fit individuals. These observations were supported by three independent studies (41, 51, 120), which observed that volition fatigue occurs at brain temperatures of 40°C in an animal model, despite various starting temperatures.

The important finding from the collective evidence provided by studies that examine the CIT model is that no animal or human died or reached catastrophic failure as a result of attaining the critical internal temperature. These observations would then argue in favour of a “safety switch” that, when “tripped”, would cause voluntary cessation of exercise (22). Mechanistically, the attainment of the CIT and subsequent volitional fatigue, immediately eliminates the metabolic heat production caused by working musculature and therefore, slows the rise in core temperature.

Pacing Strategies

The importance of regulating work rate during athletic competition has been highlighted in numerous scientific studies (39, 40), which further supported the emergence of the CGM (103). Most athletic competitions are of a closed loop design (102), whereby athletes aim to complete a required distance as quickly as possible. Competitions can be structured as either ‘head to head’ competitions or ‘against the clock’ races (40), each requiring unique pacing strategies. In head to head competition, a winning performance must only be marginally superior to that of other competitors, whereas time trial performances require athletes to exhaust the physiological capacity of their body. To appropriately examine the physiological capacity of the human body, ‘against the clock’ or time trial paces will be examined in further detail.

Abbiss et al. (2) published an excellent review summarizing pacing strategies adopted during athletic competitions. Using mathematical modelling techniques, researchers have classified pacing strategies based on power/velocity and force/time relationships into the following profiles: negative pacing, 'all out' pacing, positive pacing, even pacing, parabolic-shaped pacing, and variable pacing. Further examination of pacing strategies may provide insight into the regulation of exercise intensity, either from a central perspective or from a peripheral model.

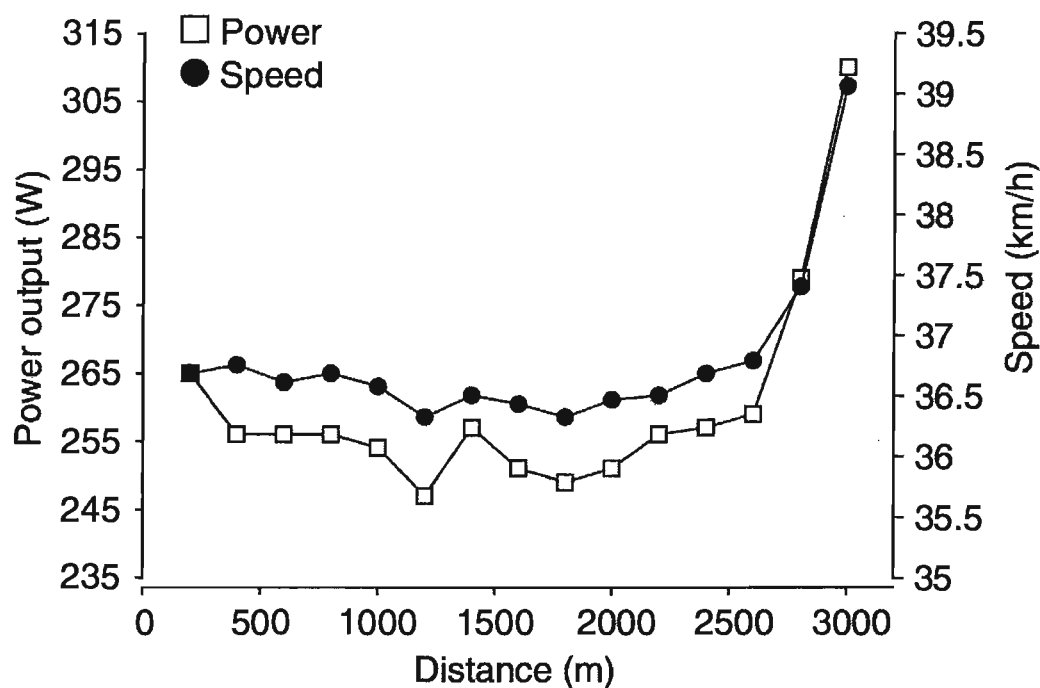
Negative Pacing

Negative pacing strategies are characterized by a gradual increase in speed throughout the duration of the competition (Figure 3) (2). Research indicates that this method of pacing reduces the rate of carbohydrate depletion (3), decreases oxygen consumption (100), and minimizes metabolite accumulation during the initial periods of the competition. Compared to self-paced exercise trials, participants who were forced to adopt a negative pacing resulted in significantly lower blood lactate levels and improved performance during a 20-km cycling time trial (76). This strategy is often characterized by increases in motor unit activation and a subsequent end-spurt towards the end of the athletic competition.

This pacing strategy supports the CGM, as work rate is regulated throughout the duration of exercise. Noakes et al. (85) suggest that initial pace is selected within minutes of exercise initiation and subsequent exercise intensity regulated to avoid catastrophic physiological failure. The gradual increase in speed observed during negative pacing can be considered as the body utilizing its physiological reserve capacity; however, this

cannot be classified as an end-spurt. Future research is required to examine the regulation of exercise during negative pacing strategies.

Figure 3. Negative pacing strategy during 3000 meter track cycling event (2).

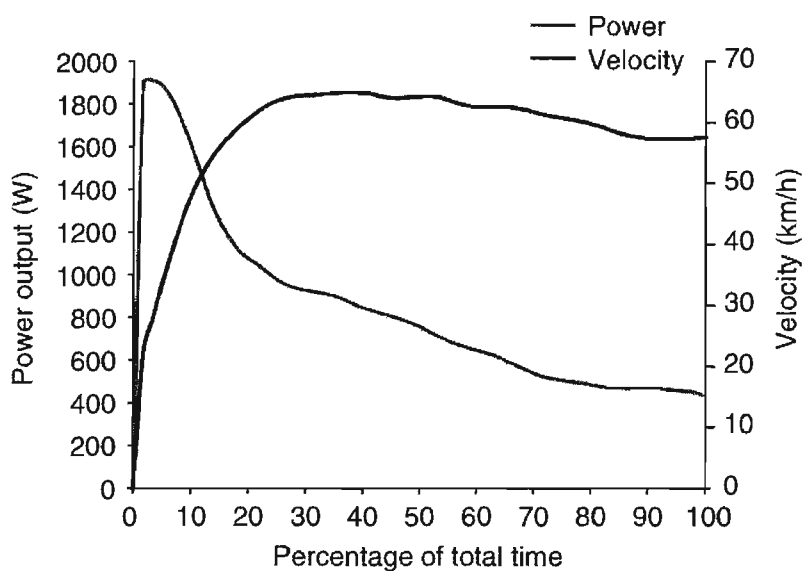


'All-Out' Pacing

When the most important aspect of an athletic competition is the initial acceleration phase, athletes may opt for an 'all out' pacing strategy (Figure 4) (2). Examples of 'all out' pacing are found in the sprint disciplines, such as the 100 m and 200 m track and field events, and are characterized by 20-25% of total energy expenditure dedicated to create kinetic energy and therefore, accelerate the body from rest. Once the athlete has accelerated to peak velocity, a gradual reduction in speed is often observed, suggesting that this pacing strategy should only be utilized during events of shorter duration (97).

Given the characteristics of this pacing strategy, it is unlikely that any central limitations of exercise intensity occur during sprint distance competitions. It is plausible that the human body has a physiological capacity to withstand catastrophic failure during exercise of short duration, thus mitigating the need for a central governor in these instances. Perhaps in an event of this duration, exercise capacity is dictated by peripheral mechanisms instead. Keller et al. (65) indicate that there are physiological limitations to exercise capacity in races of this discipline, evidence that would support a peripheral limitation on physical performance. Given this information, a question that must be addressed is: does the central governor depend on the length/intensity of exercise duration?

Figure 4. All out pacing strategy during 1000-meter track cycling event (2).

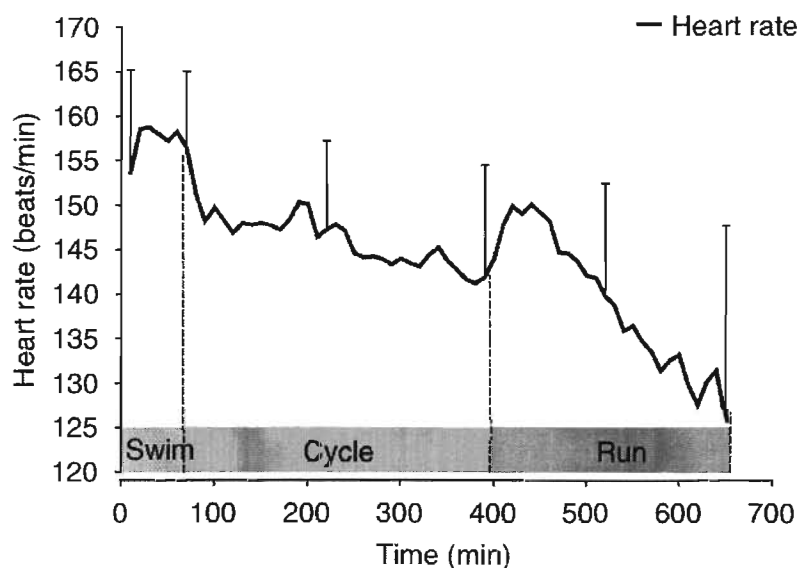


Positive Pacing

In direct contrast to negative pacing, positive pacing strategies are characterized by a gradual decrease in speed throughout the competition (Figure 5) (2). Trends indicate

that elite athletes who compete in middle distance races, such as the 400m and 800m track and field events, adopt a positive pacing strategy, which results in a progressive decrease in their speed progressively throughout the race. During such competitions, it is found that athletes obtain significantly higher $\dot{V}O_2$ values (100), compared to even pacing strategies. Furthermore, positive pacing strategies have resulted in an increased accumulation of metabolites and higher ratings of perceived exertion, therefore causing a reduction in exercise intensity. Tucker et al. (113) proposed this theory as they observed that power output decreased significantly while cycling at a set rating of perceived exertion, which ultimately resulted in substantial glycogen depletion, altered substrate utilization, and neuromuscular fatigue.

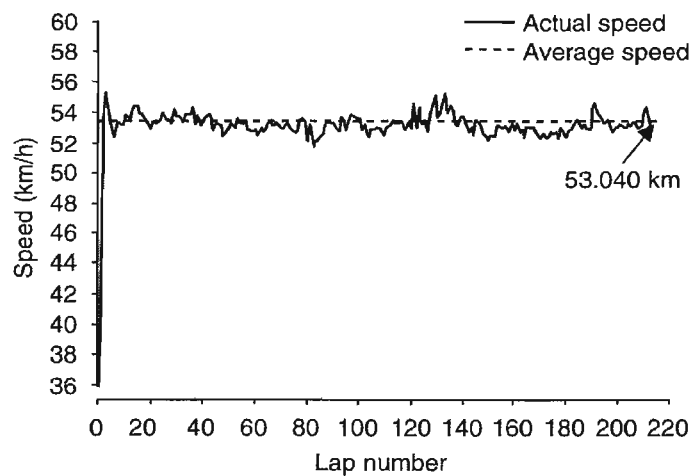
Figure 5. Positive pacing strategy during athletic competition (2).



Even Pacing

During competitions of prolonged durations, the importance of the acceleration phase is diminished due to its small contribution to overall exercise time (Figure 6) (2). In such situations, athletes are likely to adopt an even pacing strategy, which is characterized by minimal deviation in pace from mean values. Under controlled laboratory conditions, even pacing strategies prove to be optimal for exercise lasting longer than two minutes. This is emphasized by the analysis of world record performance in one hour track cycling, which indicates that each individual lap speed deviated very little from the overall average speed (93). Theoretical calculations of critical power models and mathematical laws of motion support this observation, as performance will drop if exercise intensity falls below critical power or physiological threshold. Furthermore, periodic accelerations during competition require significant force to overcome fluid resistive forces (such as air resistance), and therefore less energy is transferred into forward motion (122).

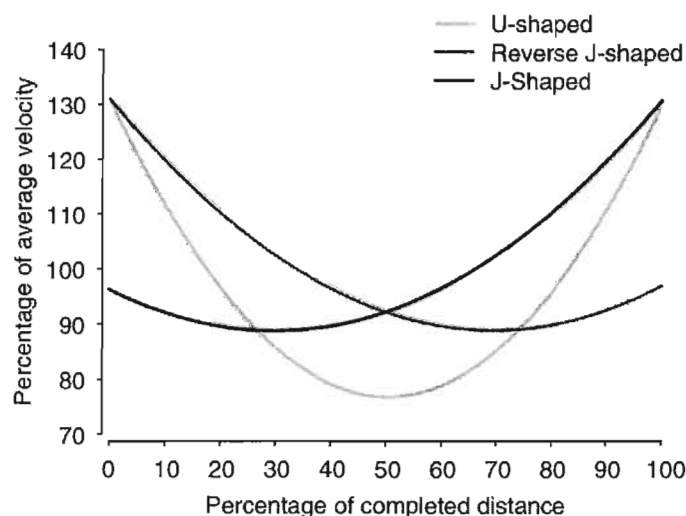
Figure 6. Even pacing strategy during pursuit cycling (2).



Parabolic-Shaped Pacing

Major technological advances in sporting equipment have allowed researches to quantify pacing strategies continuously throughout an exercise period (Figure 7) (2). A detailed observation of power output during cycling indicates that many athletes reduce speed throughout the competition until the latter portions of the event, where an increase in pace is observed. The relationship that is observed can be described as a J-shaped, reverse J-shaped, or U-shaped pacing strategy (44). Such pacing strategies have been observed in many endurance disciplines, such as rowing and cycling. Analysis of rowing performance indicates that initial 500 m split times are fastest, followed by slower pace in subsequent 1000 m section, followed by an increase in pace during the final 500 m of the race (44). Furthermore, Tucker et al. (113) observed a parabolic pacing strategy during 20 km cycling time trial, as pacing initially decreased to prevent excessive hyperthermia, followed by an 'end-spurt' during the final 5% of the trial.

Figure 7. Parabolic shaped (U-shaped, reverse J-shaped, and J-shaped) pacing strategies during athletic competition (2).



Observations of a parabolic-shaped pacing strategy are supported by the CGM, as it appears that pacing strategies decrease throughout the duration of the event, then followed by an end-spurt. It is hypothesized that the initial decrease in pace occurs to prevent excessive hyperthermia and peripheral fatigue; however, an increase in effort is possible during the final stages of competition due to a physiological reserve capacity. The psychological/motivational model can also explain these observations, as research indicates that proximity to completion of exercise may evoke an increase in physiological capacity.

Variable Pacing

Laboratory based observations of pacing strategies must be interpreted with caution as trials are conducted in a tightly controlled environment. The majority of competitions feature a dynamic environment, with constant fluctuations in race duration, course geography, and environmental conditions (2). This ultimately has an effect on pace selection throughout the competition. Field based observations of pacing selection indicate that most athletes adopt a variable pacing strategy during competition. Swain et al. (106) examined power output continuously during a cycling race and found that cycling time was superior when power output was higher on the uphill sections. It was theorized that, given the majority of time was spent riding uphill (opposed to downhill), higher power outputs while riding uphill will help maintain a more constant pace, and therefore, improve overall performance time (106). Although large accelerations during an event are inefficient due to fluid resistive forces, 5% fluctuations in power output have little effect on heart rate, \dot{V}_{O_2} , blood lactate levels, and RPE (7, 74).

Prior Experience, Feedback and Pacing Strategies

The influence of previous experience and knowledge of results during athletic competition has a profound effect on the development of a pacing strategy. The CGM suggests that, in part, previous experience modulate the centrally located controller, and therefore regulate the skeletal muscle activation during exercise (73). Specifically, previous experiences in similar events and motivation levels contribute to psychologically influence pacing strategies. Additionally, the influence of external feedback, for example devices that display heart rate, power output or speed, may supplement afferent feedback from physiological systems during exercise and cause an athlete to modify his or her pacing strategy (5). These variables create an inherent difficulty when examining the influence of central fatigue during exercise and must be addressed in the design of experimental protocols.

Prior Experience

The importance of prior experience during athletic competition is a fundamental construct of the CGM (103). It has been observed that experienced athletes tend to adopt a more appropriate pacing strategy than novice athletes, allowing them to complete an athletic event in the fastest time possible, without experiencing excessive fatigue. Athletes develop a performance template based on their previous training experience, and this information will modulate subsequent performances.

The learning effect caused by repeated performance is illustrated in a recent study by Mauger et al. (78). Athletes were separated into two groups. The control group was told that they were completing four 4-km time trials, they were notified once they completed each kilometre, The experimental group was told that they were completing

four time trials of the same distance, but did not receive any feedback. Results indicate that subjects in the experimental group gradually improved their performance with each successive time trial, whereas the control group maintained a consistent performance throughout the experiment. The more compelling finding is that the experimental group was able to develop a similar pacing strategy to the control group after completing one time trial; all subsequent time trials were not significantly different between the two groups. This finding highlights the rapid integration of previous experience into the development of an athlete's pacing strategy.

Presence of an end-spurt

As previously discussed, the conscious up-regulation of exercise intensity during the final stages of competition, or the end-spurt phenomenon, occurs regularly during self-paced exercise. In the context of pacing strategy regulation, the conscious perception of task completion will cause an athlete to increase their work output, therefore overriding the afferent feedback from the physiological systems. The end-spurt phenomenon emphasizes the need to blind subjects to the task requirements when examining the mechanism(s) that regulate exercise intensity during environmental stress. Distance or performance feedback during a trial may result in an up-regulation of exercise intensity, which means that the resultant change in work output cannot be directly attributed to the environmental conditions.

A-B-A-B Manipulation

The A-B-A-B manipulation is an experimental design used in single case studies, which examines the effect of a stimulus response (the 'B' manipulation) compared to a baseline (the 'A' manipulation) (8). This design has been widely accepted in the field of

psychology as it is commonly used to examine behavioural change (53) and furthermore, this technique has been utilized with positive results in recent human physiology research (38). The unique strength of the A-B-A-B manipulation is that it provides the opportunity to examine the effect(s) of a treatment variable on two occasions within a single case or trial (i.e., both the presence and the subsequent removal of a stimulus), therefore increasing the inferential power of the data (8). Although the potential for a carry-over effect between 'A' and 'B' conditions does exist, the purpose of the A-B-A design is to determine the temporal association between two time-series variables, not the magnitude of change.

A-B-A-B manipulations in behavioural studies research

Hall et al. conducted one of the first documented studies (53) that utilized the A-B-A-B experimental design while examining "talking-out" behaviours of young adolescents. Initially, baseline measurements were taken of the students "talking-out" behaviours while the teacher responded naturally to these disruptive actions. This was considered the first 'A' manipulation of the experimental design. The following time period, the teacher was instructed not to react to the student's outbursts, which was considered the treatment, or 'B' manipulation. This process was repeated in the same A-B fashion, alternating between baseline and treatment conditions. At the conclusion of the study, it was found that the withdrawal of attention significantly influenced the disruptive behaviour as illustrated by a high frequency of talking out behaviours during baseline periods, and a low frequency of talking out behaviours during treatment periods.

A-B-A-B manipulations in human physiology research

More recently, Flouris and Cheung (38) examined the effect of rapid changes in thermal balance on cold-induced vasodilatation, using an A-B-A-B experimental design. To examine this phenomenon, participants were submerged in warm (condition A), then cold (condition B) water, which was repeated, creating the A-B-A-B manipulation. During each manipulation, the hands were exposed to 0°C air to examine the peripheral hemodynamic response. The results of this study indicate that cold induced vasodilatation occurs only during the 'B' or cold condition, clearly illustrating the distinct differences between the two conditions. One of the unique aspects of the Flouris and Cheung study (38) is that it successfully utilizes the A-B-A-B manipulation to examine a treatment condition in the field of human physiology. The success of this experimental design indicates that an A-B-A-B manipulation may be suitable for future research in human physiology.

Estimation of Body Temperature: Thermometry versus Calorimetry

The maintenance of body temperature (T_b) around 37°C requires an equilibrium between metabolic heat production and the transfer of heat between an individual and the environment. This dynamic relationship, known as body heat storage (S), can be mathematically derived using an intricate apparatus, such as whole-body direct calorimetry or partitional calorimetric techniques. Direct calorimetry provides the most accurate measure of S and requires the monitoring of volume flow and change in temperature around a sealed chamber. Partitional calorimetry provides an indirect measure of S by measuring and calculating each component of the heat balance equation as a separate entity (21). Given the complexity of calorimetric measurements,

thermometric techniques are often employed as S can be approximated through the change in mean body temperature, body mass, and the specific heat of the body's tissues. While advantageous in being relatively simple to perform, calculations of S through thermometric techniques have been criticised in recent literature (62, 117) as it often underestimates T_b during exercise, and as stress the importance of using calorimetric techniques during such situations.

Evidence for the underestimation of S using thermometric techniques is provided by Jay and Kenny (62) in their recent deconstruction of data supporting and anticipatory regulation of exercise intensity (113). Using the two-compartment thermometry model, S of -1420 W was calculated during the first minute of exercise based on a power output of 245 W, a T_{am} of 15.1°C, and a wind speed of 2.8 m s⁻¹. Given that cycling efficiency is approximately 29%, it is estimated that metabolic heat production during the first minute of exercise was 625 W, according to heat balance calculations. Since sweating and peripheral vasodilatation heat loss mechanisms are negligible during the first minute of exercise, the 2045 W of heat loss required to achieve the reported S must be accounted for by non-evaporative heat loss. However, conservative calculations of dry heat exchange indicate a maximum of 570 W of heat loss is possible based on the environmental conditions. Therefore, the 675 W discrepancy between the calorimetric and thermometric calculations emphasises the inadequacies of the two-compartment model of thermometry (62).

Partitional Calorimetry

Calorimetry, by definition, is the quantification of the rate at which energy is transferred from the body to the environment, or vice versa (94). Partitional calorimetry,

therefore, is the technique of examining heat production and dissipation through calculating each individual avenue of heat exchange (conduction, convection, evaporation and radiation). This technique of measuring S is advantageous as it allows for precise measurements in a variety of situations, including extreme temperatures, water immersion and exercise; however, lengthy exposure times for both calorimetric and thermometric techniques are required to negate the effects of variations in hemodynamics and overall thermal balance (55, 104).

Using partitionial calorimetry, S (in $\text{W}\cdot\text{m}^{-2}$) can be calculated using the heat balance equation:

$$S = M \pm W \pm E_{\text{res}} \pm C_{\text{res}} \pm E_{\text{sk}} \pm K \pm C \pm R$$

where M represents metabolic heat production caused by the mechanical work of the body (W); E_{res} and C_{res} represent the evaporative and convective heat exchange through respiration; E_{sk} represents the evaporative heat loss from the skin; and K , C and R , represent the non-evaporative heat loss from the skin in the form of conduction, convection and radiation, respectively. Heat storage is usually expressed relative to the body surface area of the individual and can be calculated using the following formula (31):

$$A_D = 0.007184 \cdot \text{Weight}^{0.425} \cdot \text{Height}^{0.725}$$

Thermometry

As an alternative to calorimetric techniques, thermometry estimates S under the assumption that the change in body heat content is the product of the change in mean body temperature ($\Delta \bar{T}_b$), body mass, and the mean specific heat content of the body's tissues. Accordingly, S can be using the following thermometric equation (17):

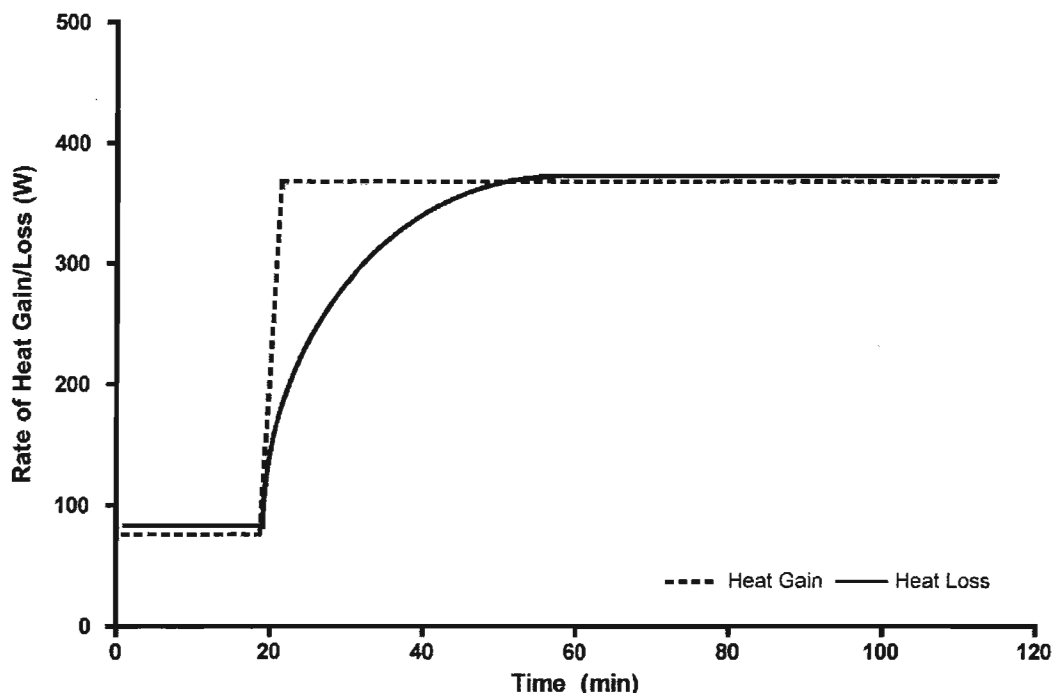
$$S = 3.47 \cdot m_b \cdot \Delta \bar{T}_b$$

where 3.47 represents the mean specific heat content of the body's tissue ($\text{kJ} \cdot \text{kg}^{-1} \cdot ^\circ\text{C}^{-1}$), m_b is body mass, and $\Delta \bar{T}_b$ is the change in body temperature over a given period of time. \bar{T}_b is calculated using the following weighted “core” and “shell” equation (54):

$$\bar{T}_b = (0.67 \cdot T_{re}) + (0.33 \cdot \bar{T}_{sk})$$

Although the simplicity of thermometry is appealing, recent examination of the two-compartment model suggests that a weighted sum of T_{re} and T_{sk} underestimates S during exercise. The onset of exercise causes an immediate increase in heat production due to the energy demands of mechanical work; however, heat loss mechanisms are slower to respond. This discrepancy creates a positive heat storage ($S > 0$), and therefore an immediate increase in T_{re} . Heat storage will only reach equilibrium once sweating and cutaneous vasodilatation mechanisms are initiated and compensate for the increase in heat production (62).

Figure 8. The rate of heat gain and loss during steady state exercise in a thermo-neutral environment (Jay et al., 2007)



A recent comparison (61) of the two- (shell and core) and the three-compartment (shell, core and muscle) thermometry models to direct calorimetry further emphasizes the inadequacies of thermometric techniques. It was found that, while cycling at 40% of $V_{O_2 \max}$, the two-compartment model under-estimated S in 24°C and 35°C by 15.5% and 35.5%, respectively, whereas the three-compartment model did not exhibit any significant bias. It was hypothesized that, during exercise, active muscle is a primary source of heat production while inactive muscle acts as a heat sink. Considering that muscle mass accounts for approximately 40% of total body mass, a thermometry model incorporating T_{sk} and T_c exclusively, does not truly reflect T_b . These findings suggest that accounting of the influence of muscle temperature can minimize the errors observed by the two-

compartment model of thermometry; however, calorimetric techniques remain the uncontested gold standard in quantifying \dot{V}_{O_2} .

Summary

Two conflicting models of fatigue have been developed to explain volitional fatigue during prolonged exercise in which exercise duration is greater than 2 minutes and requires an even distribution of energy demands (30). The CAM proposes that the cardiovascular system ultimately limits exercise capacity (13, 14), whereas the CGM claims that the brain regulates muscle activation to protect the body from physiological collapse (103). While both models have their merits, the mechanism(s) that ultimately regulates exercise intensity remains equivocal. Furthermore, the CGM proposes a theory that is logical in that the human brain is capable of dictating muscular effort; however, adequate evidence to support this model has yet to be provided. Future research using methodologies, such as partitioned calorimetry, single-case experimental designs, and the use of blinding and deception may provide more insight into the mechanism(s) that ultimately regulates voluntary pacing strategies and differentiate between these two models.

Methods

Subjects

The experimental protocol and instrumentation conformed to the standards set by the Declaration of Helsinki and was approved by the Research Ethics Board of Brock University. Twenty healthy participants (fifteen male and five female) were recruited from the local cycling club and University community and provided initial consent prior to the experiment. The mean (\pm SD) age, height, weight, body fat percentage, maximal oxygen uptake ($\text{VO}_{2\text{ peak}}$) and peak power output (PPO) of all participants was 33.7 ± 14 years, 174.7 ± 10.2 cm, 73.1 ± 13.5 kg, $12.77 \pm 6.1\%$, $56.8 \pm 9.3 \text{ ml} \cdot \text{kg}^{-1}$, and 329 ± 73 W, respectively.

Experimental Design

Each participant was required to report to the laboratory on three separate occasions separated by at least 48 hours. Twenty-four hours prior to each experimental session, participants were asked to refrain from strenuous exercise and the consumption of caffeine or alcohol. The first experimental session consisted of preliminary anthropometric testing. The second session was considered a familiarization session, which allowed participants to become accustomed to measurement techniques and the experimental protocol. The third session was the experimental trial, which was conducted in a controlled environmental chamber.

Deception and Blinding

To ensure ecological validity within the experimental design, participants were deceived to the true purpose of the experiment and were blinded to the presence of an ambient temperature manipulation. Upon recruitment, participants were informed that the title of the project was *"Power output variability while cycling at a constant rate of perceived exertion"* and were told to maintain a constant perception of effort by adjusting the workload of the ergometer if needed, with the overall goal of maintaining as steady a pace as possible. Following the completion of data collection, all participants were informed to the presence of a change in ambient temperature and were enlightened to the necessity of deception. Once this information was disclosed, participants reviewed a document outlining the true purpose and methodology of the study, then provided consent, allowing the use of their data in the final analysis.

Preliminary Testing

Maximal aerobic capacity ($\dot{V}_{O_{2max}}$) and peak power output (PPO) were quantified during the initial preliminary testing session. Participants began by completing a 5-minute warm-up on an electronically braked cycle ergometer. Males performed a 5-minute warm-up at 100 watts, followed by 25 watt increments in workload every minute until exhaustion, while females performed a 5-minute warm-up at 50 watts, with 20 watt increments in workload every minute until exhaustion. PPO was recorded as the highest level of power output achieved during the last one-minute interval. Body fat percentage was measured using the skin fold measurement technique and calculated using the Jackson and Pollock equation (59, 60).

Following the initial anthropometric testing session, participants reported to the lab for the familiarization session in which they completed a protocol that was identical to that of the experimental session. In addition to the benefits of becoming accustomed to the experimental procedures, anecdotal evidence suggests that previous experience is necessary to ride at a constant RPE without significant fluctuations in power output.

Experimental Protocol

All experimental trials were conducted in a controlled environmental chamber capable of temperature control within 0.5°C across a range from -30 to $+50^{\circ}\text{C}$ (Can-Trol Environmental Systems, Mississauga, Ontario). Throughout the experimental protocol, ambient temperature was secretly manipulated at 20-minute intervals in an A-B-A fashion from 20°C (the 'A' manipulation) to 35°C (the 'B' manipulation), then returning to 20°C . Relative humidity was maintained at 40% throughout both manipulations. Participants cycled for a total of 60 minutes on a cycle ergometer (Pro 300PT, Saris Cycling Group, Madison, Wisconsin) while maintaining a rating of perceived exertion (RPE) of 14, which corresponds to an intensity between 'somewhat hard' and 'hard' (12). The use of a cycle ergometer allowed for an open mode of exercise in which participants had free control of both resistance and cadence, and therefore power output. The main display monitor was covered from both the subject and the experimenter during the trial to remove feedback and experimental bias, and the only feedback the participant received during the experimental trial was a reminder to maintain an RPE of 14 at approximately 5 min intervals.

Instrumentation

Height (cm) and weight (kg) was measured prior to each session using standard laboratory equipment, while euhydration, defined as a urine specific gravity of 1.02 or less, was assessed using a refractometer (Atago, PAL-10S, USA). Throughout each trial, rectal temperature was measured using a thin and flexible core temperature thermistor (Mon-A-Therm Core, Mallinkrodt Medical, St Louis, MO), inserted 15 cm beyond the anal sphincter. Metabolite accumulation was quantified using a handheld blood lactate monitor (LactatePro, Kyoto, Japan) using standard laboratory procedures. Heart rate and heart rate variability were measured throughout each trial using a Polar RS800CX heart rate monitor (Polar Electro, Kempele, Finland), to quantify cardiovascular strain and autonomic control of cardiovascular function.

Heat Storage Measurements and Calculations

Residual body heat storage was measured using partitioned calorimetric techniques, and mean skin temperature was calculated using a seven point weighted averages equation, as described by Hardy and DuBois (54). Heat flux was quantified using heat flow transducers (Concept Engineering, Old Saybrook, Connecticut) placed on the forehead, abdominal, forearm, hand, quadriceps, shin and foot surfaces (54). Humidity at the surface of the skin was measured using small humidity probes (HMP50 RH/T, Vaisala Inc., Vantaa, Finland), taped parallel to the surface of the skin of the upper back, abdomen, and upper thigh. Metabolic data were collected using open-circuit spirometry (Moxus, AEI Technologies, Naperville, Illinois) to determine oxygen uptake and ventilation data during exercise

The dynamic equilibrium of core body temperature, or the rate of heat storage (S), was calculated using the following heat balance equation:

$$S = M \pm W \pm E_{res} \pm C_{res} \pm E_{sk} \pm K \pm C \pm R$$

where M represents the heat created by metabolism, specifically the transport of oxygen throughout the body, and was calculated using the following equation (42):

$$M = [(0.23 \cdot RQ + 0.77) \cdot 21.14] \left(60 \cdot \dot{V}_{O_2} \right) \cdot A_D$$

where RQ represents the respiratory quotient, and A_D is the body surface area, which was calculated by (31):

$$A_D = 0.007184 \cdot Weight^{0.425} \cdot Height^{0.725}$$

W is the release of heat though the mechanical work of the human body. E_{res} represents the transfer of heat though evaporative process of respiration and C_{res} represents the transfer of heat through convective processes of respiration, and was modelled by the following equations, respectively (36):

$$E_{res} = 0.0023 \cdot M \cdot (6.51 - P_a)$$

$$C_{res} = 0.0014 \cdot M \cdot (37 - T_a)$$

Evaporative heat loss through the skin, as indicated by E_{sk} , was modelled by the following equation (70):

$$E_{sk} = h_e \left(\phi_s \cdot p_s - p_a \right) \left(1 + \left(\frac{RT}{\lambda} \right) \ln \left(\frac{1}{\phi_s} \right) \right)$$

where h_e represents the coefficient for heat exchange by evaporation ($W \cdot m^{-2}$), ϕ_s represents the relative humidity at the surface of the skin, P_s represents the saturated

water vapour pressure at skin temperature (KPa), P_a represents the partial pressure of water vapour in ambient air (KPa), T represents skin temperature ($^{\circ}\text{C}$), and λ represents the latent heat of vaporization (J/g). The partial pressure of water vapour can be calculated by Antoine's equation as follows:

$$P_{sa} = \exp\left(18.956 - \frac{4030.18}{t + 235}\right)$$

Thermal resistance is represented by R and is modelled by the following equation:

$$R = \frac{1}{h} = \frac{1}{h_r + h_c}$$

where h_r represents the radiative heat transfer coefficient, and h_c represents the convective heat transfer coefficient. Heat transfer coefficients were recently determined by Kurazumi et al. (72) using thermal manikins, and are listed below for convection and radiation, respectively:

$$h_c = 1.175\Delta T^{0.351}$$

$$h_r = 3.871$$

Statistical Analysis

To examine any possible synchronicity between changes in PO and thermo-physiological status, Auto-Regressive Integrated Moving Averages (ARIMA) time series analysis was employed. ARIMA combines three statistical processes, autoregression, integration/differencing, and moving averages, and mathematically describes association in a disturbance in one time series (in this case, thermo-physiological status) and the possible perturbation in a second time series (power output). Using specific model

building procedures, an appropriate ARIMA[a,d,q] model can be specified based on the calculated autoregression (a), differencing (d) and moving averages (q) integers. Given the stringent nature of an ARIMA analysis, data must be collected in time series with adequate resolution to construct a viable model.

To remove any inter-individual variability, PO, T_{sk} , HS, and T_{bd} were transformed from absolute values to change from baseline values for each individual participant. The changes in PO (ΔPO) values were determined by comparing each value to the average PO as calculated during the first 20 minute 'A' manipulation. The rate of change in heat storage (ΔS) was calculated by subtracting an instantaneous value of heat storage by the preceding value ($\Delta S = \Delta S_t - \Delta S_{t-1}$). The change in T_{sk} (ΔT_{sk}), and T_{bd} (ΔT_{bd}), was calculated by normalizing each value to initial T_{sk} , and T_{bd} , with measurements being taken during the first 30-seconds of the experimental trial.

All continuous variables were smoothed into 30-second averages to analyze any possible synchronicity among variables. Preliminary analysis consisted of graphing all time series data (ΔPO , ΔT_{sk} , ΔT_{bd} , and SR) against time to determine any systematic seasonal patterns as illustrated by rhythmic fluctuations in a variable. An autocorrelation analysis was conducted, indicating a progressive decline in the autocorrelation scores throughout the time series and therefore, that the time series is non-stationary. To comply with the ARIMA assumptions, the data were transformed using differencing procedures, rendering the data stationary. Subsequently, the correlation between the two time series variables was analyzed using a stationary r^2 , given the stationary nature of the data.

To analyse potential synchronicity between PO (the dependent variable) and ΔT_{sk} , ΔT_{bd} , and SR (the independent variable), an Auto Regressive Integrated Moving Average

(ARIMA) model was constructed based on the combined time series data from all participants. Therefore, ARIMA was used to determine whether changes in PO were caused by any fluctuations in any thermo-physiological variables. The integrity of the ARIMA model was determined using the Ljung-Box test with a significance level of $p > 0.05$, indicating that the model was correctly specified.

Heart rate variability (HRV) was analyzed using the time-domain, SDANN method, with the standard deviation of the time delay between each successive heartbeat being calculated over a 5-minute period (96). Capillary blood lactate measurements (LA) were taken during baseline, and 5 minutes prior to the end of each manipulation (i.e., at 15, 35, and 55 minutes). Given the non-continuous nature of the HRV and BL data, separate repeated measures analysis of variance (ANOVA) and Scheffé *post-hoc* analysis were used to determine differences in autonomic nervous functioning and metabolite accumulation, respectively, between each manipulation.

All statistical analyses were conducted using SPSS 17.0 (SPSS Inc., Chicago, Illinois). Statistical significance was set at $p < 0.05$.

Results

Environmental Response

During the experimental trial, T_{am} was successfully manipulated in an A-B-A pattern from 20°C (the 'A' manipulation) to 35°C (the 'B' manipulation) then returning to 20°C (see Figure 10). After a 2-minute equilibrium period, T_{am} reached 20°C and remained at steady state for the remainder of the first 'A' manipulation. Upon initiation of the 'B' (35°C) manipulation, T_{am} increased gradually at a rate of $1.154\text{ }^{\circ}\text{C}\cdot\text{min}^{-1}$ and reached its peak approximately 38 minutes into the experimental trial. T_{am} then remained at equilibrium for the remaining 2-minutes of the 'B' manipulation. Following the initiation of the second 'A' manipulation, 40 minutes into the experimental protocol, T_{am} decreased sharply at a rate of $3.227\text{ }^{\circ}\text{C}\cdot\text{min}^{-1}$ and reached its nadir 45.5 minutes into the experimental trial. A steady state temperature of 20°C was achieved 14.5 minutes following the initiation of the second 'A' manipulation and remained at equilibrium until the completion of the experimental trial.

Behavioural Response

Figure 9 represents the individual data from one subject selected at random for PO, T_{am} , HR, S, SR, T_{sk} and T_{re} throughout the 60-minute experimental trial. The observed patterns in these figures are consistent in all 20 participants.

The main finding of the present study is that changes in ambient temperature cause fluctuations in PO. Initially, subjects exhibited a period of pacing selection in which PO fluctuated significantly during the first 10 minutes of the experimental trial.

Figure 9. Individual data selected at random for PO, T_{am} , HR, S, SR, T_{sk} and T_{re} throughout the 60-minute A-B-A trial.

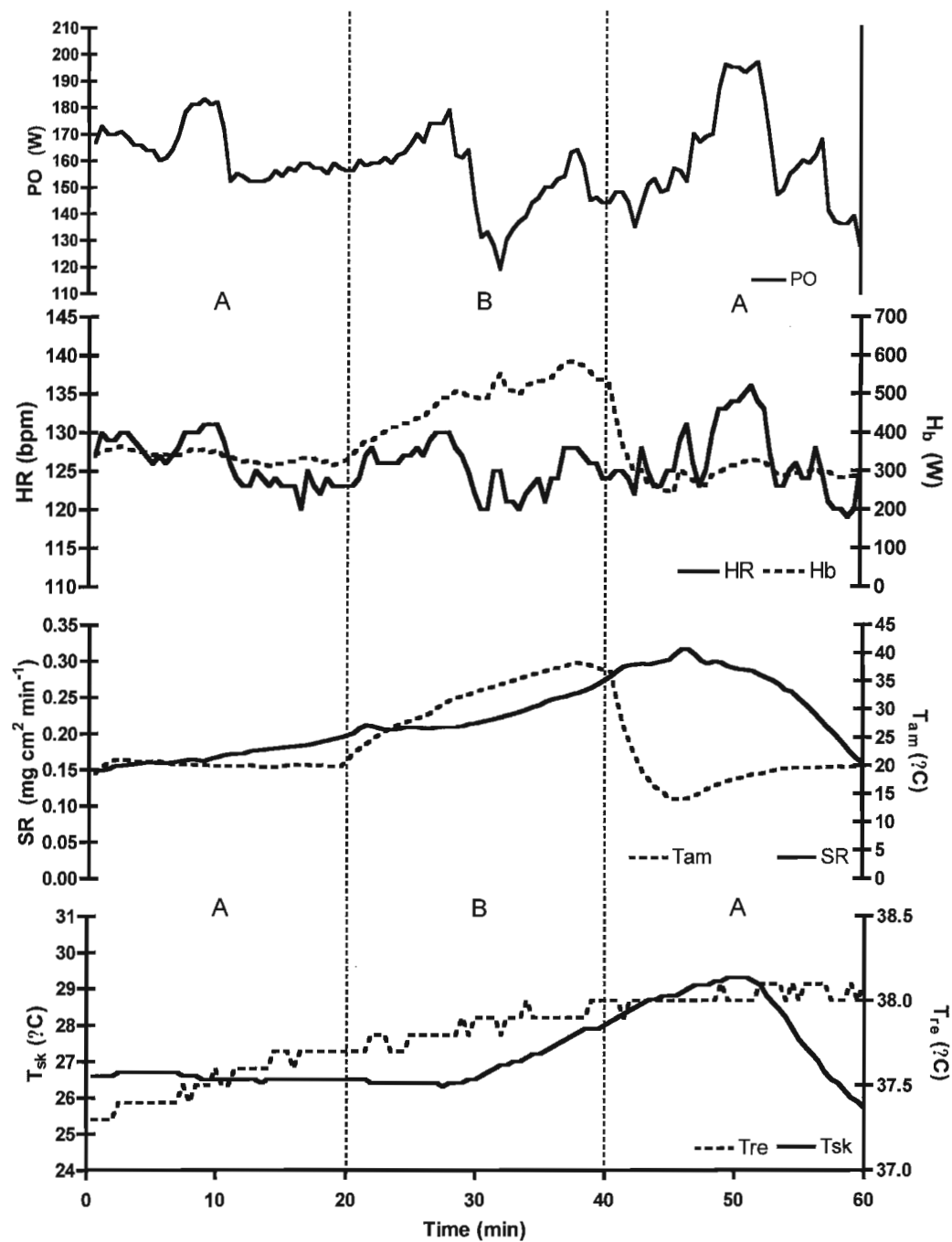
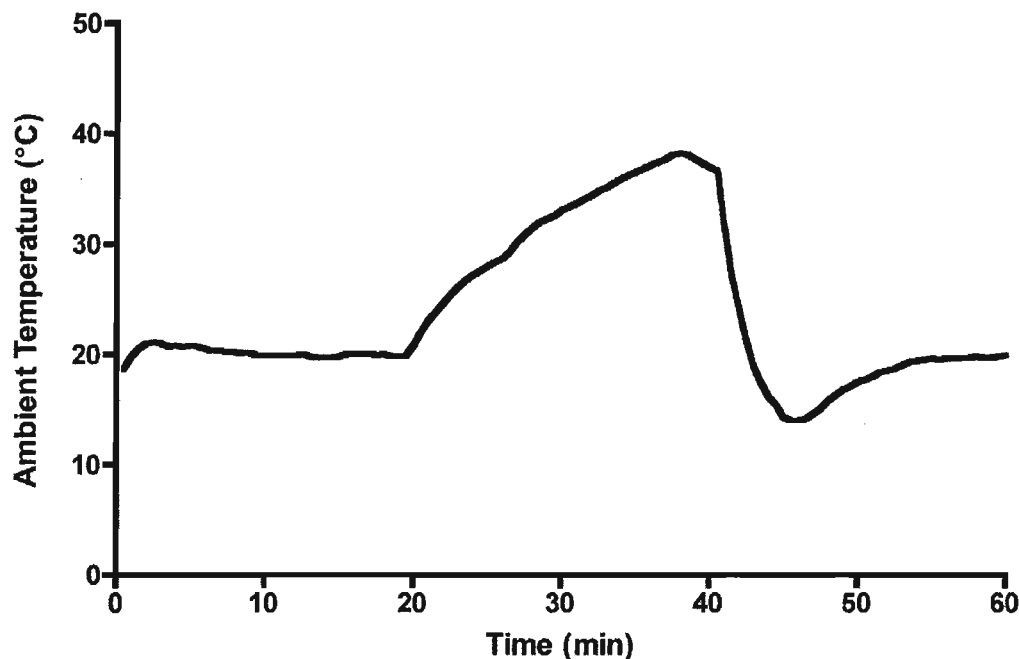


Figure 10. Change in T_{am} throughout the 60 min A-B-A manipulation.



Following this initial 10-minute period, PO remained stable at baseline values for the remainder of the first 'A' manipulation. Upon the initiation of the 'B' manipulation, participants exhibited a gradual increase in PO of approximately 15 watts above baseline values, peaking at 8 minutes following the initiation of the 'B' response. After peaking, PO decreased sharply, reaching levels approximately 44 watts below baseline values 10 minutes following the initiation of the 'B' manipulation. Participants then displayed a second period of pacing selection in which PO was highly variable, lasting for the remainder of the 'B' manipulation. In response to the second 'A' manipulation, participants exhibited an increase in PO to wattages above baseline values, reaching values of 30 watts above baseline, 10 minutes following the initiation of the second 'A' manipulation. This peak in PO was followed by a third period of pacing selection, which lasted for the remainder of the experimental trial.

Physiological Responses

Although there is an apparent behavioural response to an altered T_{am} , the present experimental design and the ARIMA analysis would suggest that perturbations in PO are not associated with changes in any of the collected thermo-physiological variables, specifically, changes in ΔT_{sk} , ΔS , ΔT_{bd} , and ΔSR . The fluctuations in the aforementioned physiological variables were examined using an ARIMA[2,0,0], with the specific autoregression, integrated and moving average integers determined using the expert modeller function of SPSS 17.0. Table 1 outlines the specific stationary r^2 values and Ljung-Box statistic associated with each predictor variable for PO.

Table 1. Independent predictor variables and associated stationary r^2 and Ljung-Box statistics resultant from the ARIMA[2,0,0] analysis.

Predictor Variable	Stationary r^2 (sig.)	Ljung-Box Statistic (sig.)
ΔT_{sk}	0.077 (0.798)	16.336 (0.430)
ΔHS	0.100 (0.091)	15.875 (0.462)
ΔT_{bd}	0.084 (0.340)	14.869 (0.534)
SR	0.077 (0.942)	16.554 (0.415)

Capillary blood lactate data are displayed in Figure 10. A significant increase in blood lactate values was observed during the 'B' manipulation ($6.8 \pm 3.6 \text{ mmol} \cdot \text{L}^{-1}$) when compared to baseline values ($p=0.001$); however, there were no significant differences between the first ($4.8 \pm 1.6 \text{ mmol} \cdot \text{L}^{-1}$) and second ($4.6 \pm 3.6 \text{ mmol} \cdot \text{L}^{-1}$) 'A' manipulations when compared to baseline values ($p = 0.264$ and 0.191 for the first and second 'A' manipulation, respectively). Non-significant increases in blood lactate values were

observed when comparing the 'B' manipulation to the first ($p=0.216$) and second ($p = 0.295$) 'A' manipulations. Heart rate variability throughout the 60-minute trial is displayed in Figure 11 and did not change significantly over time ($p > 0.05$).

Figure 11. Capillary blood lactate concentration ($\text{mmol}\cdot\text{L}^{-1}$) during baseline (time 0) and 15 minutes following the initiation of each A-B-A manipulation, at time 15, 35 and 55 minutes, respectively.

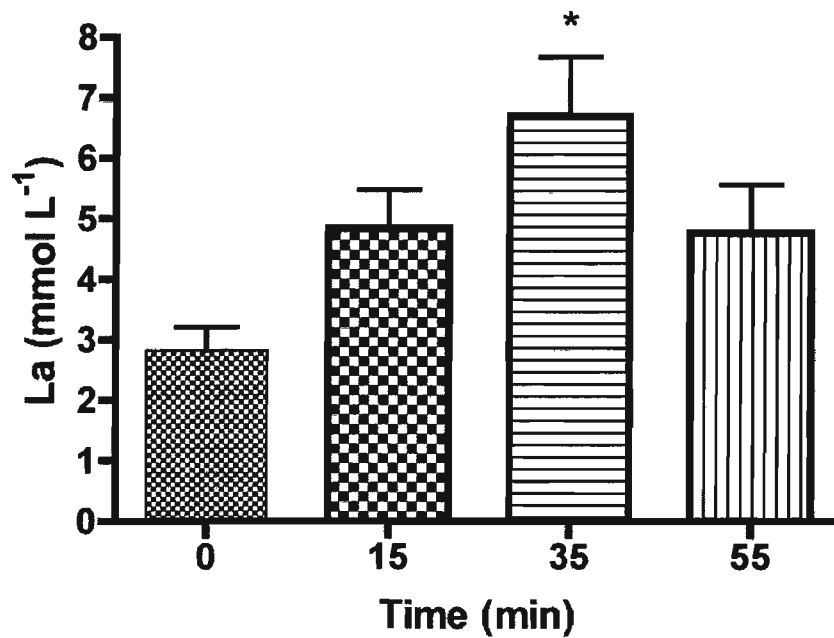


Figure 12. Change in heart rate variability (SDANN) throughout the A-B-A manipulation of T_{am} .

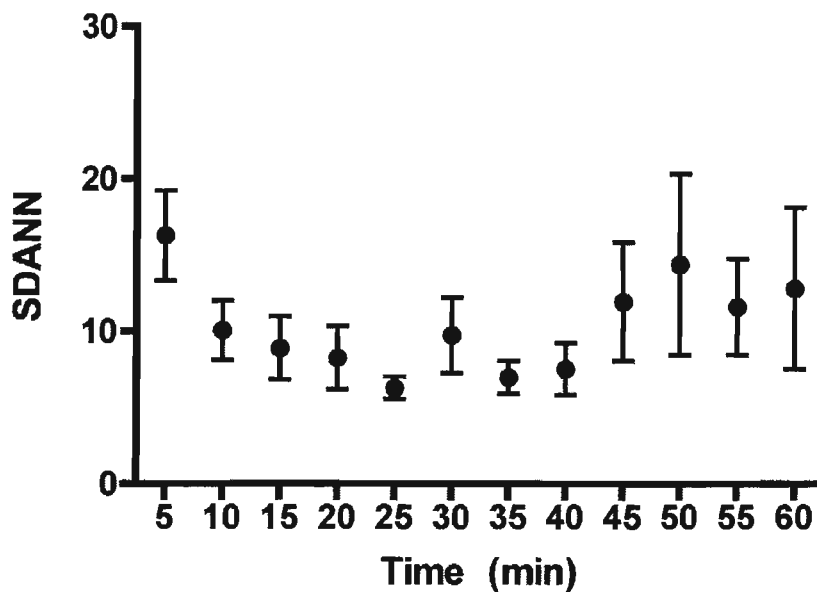
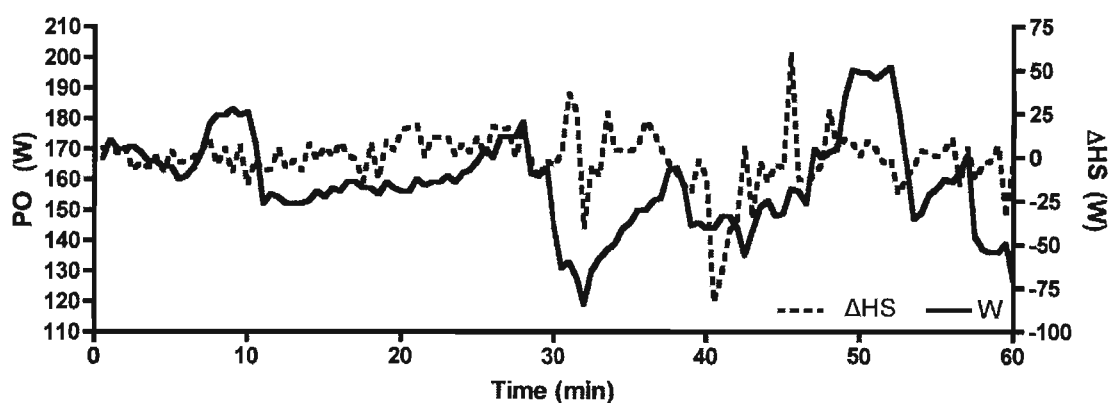


Figure 13. The change in the rate of heat storage and power output throughout the A-B-A manipulation of ambient temperature.



Discussion

Presently, two distinct models of fatigue have been proposed to explain the mechanism(s) that regulate exercise intensity during heat stress. The CAM holds that the increased cutaneous vascular conductance, and the demand for nutrient supply and waste removal in the working musculature causes competition in blood flow, resulting in an impaired exercise performance (98). Although the CAM is widely accepted in the scientific community, alternative models, namely the CGM, postulate that exercise is regulated in response to ambient stress, thereby preventing catastrophic physiological failure in multiple physiological systems (113, 115). The purpose of the present study was to directly test a prediction of the CGM, specifically, that PO is down-regulated in response to increased thermal stress, by secretly manipulating T_{am} in a dynamic A-B-A manner from 20°C (the 'A' manipulation) to 35°C (the 'B' manipulation), then returning to 20°C (back to the 'A' manipulation). Participants were asked to cycle at a constant perception of effort, corresponding to 14 on the Borg Scale (12), and throughout the test were not given any feedback based on their performance. The exercise intensity and environmental stress were chosen specifically to allow teleo-anticipation without being stressful enough to cause peripheral fatigue. The blinded nature of the study and the single trial experimental design addresses inherent limitations in previous research and creates a novel method of testing central fatigue during heat stress.

The main finding of the present study is that, based on the current experimental design, changes in PO were not statistically associated with changes in any of the collected thermo-physiological variables. In response to each manipulation, either from 20°C to 35°C (A to B) or 35°C to 20°C (B to A), perturbations in PO occur; however,

this apparent behavioural response could not be explained by an anticipatory feed-forward regulation of exercise intensity based on thermal cues. These findings conflict with previous research (63, 113, 115), which suggest that exercise capacity is regulated by afferent feedback to a centrally located controller. The CGM hypothesizes that in response to heat stress, increases in heart rate and respiratory rate, along with decreases in blood glucose level (45) cause an increase in RPE. Proponents of the CGM believe that this increase in RPE mediates alterations in skeletal muscle activation and therefore, down-regulates exercise intensity (114).

Previous research supporting the CGM suggests that the inherent increase in RPE during heat stress is instigated by changes in the rate of heat storage (ΔS) (113, 115). The crux of this theory is based on the heat balance response to exercise and heat stress. The onset of exercise causes an initial increase in core temperature ($\Delta S > 0$) due to the instantaneous liberation of energy required to meet the metabolic requirements of the active musculature. Heat loss mechanisms, namely sweating and peripheral vasodilatation, are slower to respond; however, given adequate time, these alterations have the capacity to offset the rate of metabolic heat production ($\Delta S = 0$). When an athlete exercises in environments of higher ambient temperature, the transfer of heat from the environment to the body occurs primarily through non-evaporative avenues (assuming that there are no changes in relative humidity). The increase in ambient temperature causes a shift in the temperature gradient between the skin and the environment, resulting in an increased convective heat transfer to the body and therefore, an increase in core temperature ($\Delta S > 0$). Tucker et al. (113) suggested that the rate of heat storage is the critical variable in the regulation of exercise intensity during heat stress.

During the hot (35°C) trial, the higher rate of heat storage in comparison with the normal (25°C) and cool (15°C) trials resulted in the athlete exercising at a lower work output, leading to a decreased metabolic heat production. This decrease in heat production compensates for the increase in convective heat gain and therefore, maintains thermal homeostasis ($\Delta S = 0$). The present study examined the relationship between changes in PO and ΔS and found no synchronicity (stationary $r^2 = 0.100$, $p = 0.091$; Ljung-Box statistic = 15.875, $p = 0.462$) between the two variables (Figure 12). No repeatable patterns in ΔS were observed throughout the A-B-A manipulation of T_{am} . Initially, ΔS was stable until the initiation of the 'B' 35°C manipulation, where large fluctuations were observed. This evidence suggests that, during a dynamic manipulation of T_{am} , ΔS is not a critical variable, and does not contribute to the anticipatory reduction in exercise intensity.

The method of heat storage calculation may account for the contrasting findings, as thermometric calculations (110) have been criticized as being inaccurate for such dynamic settings (68). The calorimetric measurement of S , as employed in the present study, provides the most accurate quantification of changes in thermal balance during exercise. Previous research (113) in which a reduction in power output was observed in response to changes in S , utilized thermometric calculations of residual body heat storage. The limitations of using the two-compartment (T_{re} and T_{sk}) calculation of T_{bd} and therefore, S are twofold. Primarily, estimations of S during the initial phase of exercise are drastically under-estimated due to the delayed activation of heat loss mechanisms. Additionally, the large thermal inertia of the pelvic region causes a gradual change in rectal temperature, and does not adequately reflect the minute-by-minute changes in total

body heat content. Therefore, it is plausible that the association between the rate of heat storage and power output observed in previous studies (113) was due to methodological errors, rather than changes in thermal status.

An alternative explanation for the observed decrease in work output during heat stress lies within the field of thermoregulatory behaviour. Schlader et al. (101) indicate that changes in T_{sk} drives changes in thermal comfort, and therefore initiate conscious actions to modulate one's thermal status. While exercising in the heat, it is plausible that an increased skin temperature will prompt an individual to voluntarily down-regulate power output in order to maintain ΔS . The importance of skin temperature during self-paced cycling is apparent when observing an anticipatory reduction in muscle activation while cycling in a hot environment. Tatterson et al. (109) report a 6.5% decrease in power output while cycling in 32°C (hot) when compared to 23°C (cool); however, the compelling finding was that, despite the 9°C difference in ambient temperature, core temperature remained similar. It is believed that the increase in T_{sk} during the hot condition causes a decrease in neural drive (109), and in an anticipatory fashion, protects the body from accumulating excessive heat; however, the data from the present study do not support this hypothesis.

In the present study, ARIMA analysis indicates a lack of synchronicity (stationary r -squared = 0.077, p = 0.798; Ljung-Box statistic = 16.336, p = 0.430) between the increase in skin temperature and the decrease in power output during the A-B-A manipulation of ambient temperature. These results do not support a thermo-behavioural response mediated by a change in skin temperature (101) and therefore, suggest that the behavioural response to heat stress during exercise involves a complex interplay of

psychological and physiological factors. Crewe et al. (28) suggest that changes in skin temperature do not initiate a reflexive change in power output, rather indirectly modulate power output through changes in thermal sensation, thermal comfort and ultimately, RPE. Therefore, given the multiple degrees of separation between the changes in skin temperature and the eventual behavioural response (in this case, power output), it would be illogical to expect a high degree of synchronicity between the two variables in the present study.

Alternatively, the conscious desire to maintain a constant mean body temperature during rest and exercise has been shown to initiate thermoregulatory behaviour. When allowed to freely regulate the temperature of water circulating through a liquid conditioning garment, participants chose to regulate the inflow of water temperature to maintain a constant thermal comfort, thermal sensation and mean body temperature (37). In the present study, when subjected to a dynamic manipulation of ambient temperature, changes in mean body temperature are not correlated (stationary- $r^2 = 0.084$, $p = 0.340$; Ljung-Box statistic = 14.869, $p = 0.534$) to the initiation of thermo-regulatory behaviour. Although changes in T_{bd} have been shown to initiate thermoregulatory behaviour, theoretical limitations compromise the prediction of this model when applied to exercise. While exercising in a heat stress environment, core temperature exhibits a gradual increase over time, irrespective of changes in power output. Given the high weighting coefficient associated with core temperature, it is unlikely that T_{bd} would exhibit an A-B-A response to changes in ambient temperature in the present study. As previously discussed, the limitations of thermometric body heat content measurements, specifically

in response to dynamic exercise, compromise the integrity of a T_{bd} modulated change in power output.

Proponents of the CAM may argue that the reduced power output occurring during the 35°C 'B' manipulation is caused by metabolite accumulation or altered autonomic nervous function. During periods of high intensity exercise, energy metabolism shifts from primarily aerobic to anaerobic pathways, resulting in higher accumulation of blood lactate concentration. Research (4, 74) indicates that there is a strong correlation between increases in blood lactate concentration and reductions in power output. In the present study, blood lactate concentration during the 60-minute trial illustrated a characteristic, exercise induced increase in response to the onset of exercise; however, blood lactate concentration did not fluctuate with any statistical significance during the A-B-A manipulation of ambient temperature. Furthermore, the lack of significant fluctuations in heart rate variability throughout the trial indicates no changes in autonomic nervous function. Collectively, these results indicate that there is no shift to anaerobic metabolism or changes in autonomic nervous function and therefore, do not directly support the CAM.

Unique to this study is the use of an A-B-A manipulation, in which the effect of a stimulus response (in this case, an increased T_{am} during the 'B' manipulation) on baseline values (PO during the first 'A' manipulation), followed by the removal of the stimulus (the second 'A' manipulation) was examined. The merits of this experimental design have been exploited in behavioural research (53) and more recently, have been utilized in human physiology research (38). Specifically, the inferential power of examining the stimulus and removal of a treatment variable is much greater when compared to

examining only the treatment variable itself (8). In the context of this research project, it can be concluded that fluctuations in PO occur as a result of psychological and/or physiological mechanisms, rather than through any inherent daily variability of PO.

The importance of blinding and deception is essential when testing paradigms of central fatigue. Although very insightful, previous studies (113, 115) have not addressed the psycho-physiological mechanisms that may influence pacing strategies when the presence of an environmental stressor is known. Although the mechanisms related to the perception of thermal stress and the resultant influence on exercise performance remains an intricate issue, the impact of heat stress on thermo-behaviour is undoubted. Maw et al. (79) report a higher thermal discomfort and subjective ratings of perceived exertion while cycling at a constant workload in the heat, which suggests that this psychological process modulates the resultant behavioural response. Therefore, prior knowledge of an environmental manipulation may cause subjects to adopt a pacing strategy based on the conscious knowledge of a stressor, rather than a subconscious down-regulation of exercise intensity based on afferent feedback from multiple physiological systems.

Knowledge of performance requirements has been shown to influence pacing strategies, irrespective of environmental stressors. Many studies (35, 115) have documented an increase in exercise intensity, a phenomenon known as an 'end-spurt', when in close proximity to perceived task completion (18). This apparent increase in motivation causes athletes to sub-consciously or consciously increase their power output, effectively over-riding the decision making process based on afferent feedback from their physiological systems. This study suggest that, due to the use of deception, blinding, and the 'RPE' clamp protocol, the increase in PO observed during the second 'A'

manipulation was a result of an afferent feedback modulation in power output, not psychological factors associated with the proximity to the endpoint of exercise.

Furthermore, Mauger et al. (78) demonstrate the importance of prior knowledge when developing a pacing strategy during athletic competition. When completing successive 4 km time trials, distance blinded subjects completed the required distance at a slower pace when compared to subjects who received feedback at regular intervals (the control group) with both groups improving with each successive bout. Distance blinded subjects completed the fourth time trial in similar time as control subjects, clearly illustrating a learning effect. These results indicate that when the performance requirements are not outlined and regular feedback is not provided, such as in the current experimental protocol, initial pacing strategies are regulated exclusively on afferent feedback from multiple physiological systems without the influence of any psychological confounds.

Although a dynamic A-B-A manipulation of ambient temperature does not cause a synchronized, anticipatory reduction in power output, the fundamental concept of the CGM, namely that exercise intensity is regulated prior to catastrophic physiological failure, has not been disproven here. A chief pillar of the CGM is the presence of an end-spurt during exercise; that is, a conscious up-regulation of exercise intensity when in close proximity to the end of the event. Proponents of the CGM argue that the end-spurt phenomenon disproves the CAM (87), namely that muscle activation, and consequently power output, are regulated by feedback from the fatiguing skeletal muscle (6) and therefore, does not explain the up-regulation in exercise intensity. The present study specifically eliminated previous experience and knowledge of results by blinding the

participants to the presence of an environmental manipulation and removed external feedback. This design consideration was made to specifically test the power output response to changes in ambient temperature, rather than psychological factors; however, the inherent limitation of this design is that it does not present the opportunity for the observation of an end-spurt.

Conclusions

The present study aimed to directly test a primary of the CGM, that is, exercise intensity is down regulated in anticipation of preventing catastrophic physiological collapse. When cycling at a constant RPE of 14 (12), changes in PO were observed which did not correspond to any observed perturbations in physiological homeostasis. This observation conflicts with previous research (113, 115) that supports the CGM, in that we saw no apparent anticipatory reduction in exercise intensity. Furthermore, there was no discernible synchronicity between the changes in T_{sk} and T_{bd} and any thermoregulatory behavioural response. These results suggest that the mechanism(s) that regulate power output during self-paced exercise involves a more complex interplay of psychological and physiological factors than simply an abrupt change in thermal status.

Limitations

Although the present study featured a novel research methodology that addressed many shortcomings in previous work, several inherent design limitations may confound the present results. A crucial aspect of the present design was to elicit a specific magnitude of stress, as determined by the ambient temperature (35°C) and exercise intensity (14 on the Borg Scale); however, the resultant strain may have been too stressful or not stressful enough due to individual differences. Theoretically, the Borg scale is analogous to heart rate (i.e., RPE of 14 should equate to a heart rate of 140 – 149), based on values derived from a 20 year old individual (12). Given that maximum heart rate is correlated with age, those individuals who deviate significantly in age from the “typical” values of a 20 year old may find that an RPE of 14 is too stressful for an older participant,

whereas a younger participant may not find an RPE of 14 stressful enough to elicit an anticipatory change in exercise intensity. This is because a heart rate of 140 beats per minute is a great percentage of max heart rate for older participants, relative to younger participants.

An additional limitation to the present study is the difficulty in completely blinding the subject to the manipulation in ambient temperature. The rapid, 15°C change in ambient temperature was easily sensed by all participants and therefore, may cause a down-regulation in exercise intensity based on the conscious perception of heat stress, rather than physiological afferents from the body. This potential limitation was addressed prior to the experimental sessions through specific instructions to maintain a constant perception of effort throughout the entire duration of the trial.

Future Directions

Although the present study has pioneered many methodological techniques, further research is required to fully understand the mechanism(s) that contributes to fatigue during environmental stress. To supplement the current data, electromyography (EMG) can be used to quantify the efferent information from the central controller as to determine any central regulation in exercise performance. Previous research (113, 115) indicates that reductions in EMG activity coincide with increases in ambient temperature, which supports the notion that exercise is regulated by central fatigue. Given the ambiguity of the results based on the skin blood flow data, a more thorough analysis of the hemodynamic response to exercise in the heat is needed.

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Appendix A: Research Ethics Consent Form

Informed Consent: EEL-055

Project Title: Power Output Variability While Cycling at a Constant Rate of Perceived Exertion (EEL-055)

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INVITATION

You are invited to participate in a study that involves research. The purpose of this study is to test the ability to maintain a stable power output at a constant perceived exertion during a prolonged ride. These measurements will give us information on the reliability of trained cyclists to maintain power output at a given RPE during a prolonged cycle.

WHAT'S INVOLVED

There will be a total of three sessions. The first session will test your fitness, followed by one familiarization ride, and one testing ride. Females will be tested at the follicular phase of the menstrual cycle (3-12 days following the start of menstruation). Time commitment will be approximately 4.5 hours over the three sessions, spaced out over approximately three weeks for males. Testing may be spread out over two months for females to make sure that the experimental session is done at the follicular phase of the menstrual cycle. Prior to each session, you will be asked to refrain from alcohol and/or heavy exercise for 24 hours prior to the trial and caffeine on the day of the trial. In all three sessions, you will change into your own cycling shirt and shorts. Appropriate change rooms will be provided for you to change into the required clothing. You will have free access to water throughout all sessions.

In the first session, you will have your height, weight, and the amount of body fat in your body measured. Body fat testing will be performed using skinfold callipers, which might cause a slight pinching sensation, and will be taken by someone of the same sex in a private room. You will also ride on a bicycle ergometer at gradually increasing workloads until exhaustion in order to obtain your peak power output (PPO). The total session will take about 1.5 h.

In the second session, you will practice the bike test. After 5 min of easy pedalling on the stationary bicycle, you will ride for approximately 45 minutes at an effort that feels 'somewhat hard' or 14 on the Borg RPE scale. You are free to adjust the workload of the ergometer, but will not be given feedback on the workload or how long you have been riding. You will be asked for your subjective comfort, and ratings of perceived exertion at 5 min intervals throughout the experiment. The total session will take about 1 hour.

In the experimental session, you will have your internal temperature measured by wearing a rectal temperature sensor. The rectal sensor consists of a very thin and flexible plastic tube that you insert 15 cm beyond the anus. Skin temperature and heat flow will be monitored using wires taped to various sites on your body. The electrical activity of your leg muscles will be measured by electrodes that will be taped to your thigh. Your oxygen uptake will be measured by having you breathe through a soft silicone mask. You will also be asked at different times to give your subjective feeling of your effort and also your comfort. You will wear a soft and flexible strap around your chest to measure heart rate. A technician of the same sex will be available to assist with the dressing and instrumentation. You will have small finger prick samples of blood, obtained using a sterile lancet, to measure your blood lactate. Prior to and following the cycling test, you will have your body weight measured, and will be asked to provide a small urine sample to ensure that you are properly hydrated.

In the experimental session, you will be exercising in an environmental chamber (11oC, 30% relative humidity) to control the surrounding conditions. You will have a 10 min easy warm-up, and then be asked to ride at a constant perceived effort of "14" on the 6-20 Borg scale, which is equivalent to an effort between "somewhat hard" and "hard." You will not be given feedback (e.g. heart rate, power output, time) during this test, as we are measuring the effectiveness of your self-determined ability in maintaining constant power output during prolonged exercise. The cycling session will last 90 min and the entire session will take about 2 h. You will not be able to drink water during the cycling test, but will be given as much fluid as you wish after the experiment.

POTENTIAL BENEFITS AND RISKS

Possible benefits of participation include your receiving a fitness test (peak power output) that will help you to understand your cycling fitness level. You will also receive \$50 for completion of the experiment, with payment prorated for partial completion.

There may be risks associated with participation. The exercise test is an effort that is a bit harder than a typical jog, but less than what might happen during a 10 km running race. You may feel quite tired for up to 48 h after the test. There is a very remote risk of heart attack or stroke when exercising to exhaustion, but this is minimized with the use of the health screening questionnaire. There will be at least two investigators trained in First Aid and CPR present for each experiment. The investigators will contact you later in the day following each session to check on your health status.

Experimental sessions will be terminated if:

1. Rectal temperature increases beyond 39.5oC.
2. Heart rate has risen above 95% of its maximum (220-age) for 3 min.
3. Dizziness or nausea precludes further experimentation.
4. You decide, for any reason, to end the experiment.
5. The investigators determine that the subject is unable/unfit to continue.

Insertion of the flexible rectal probe may cause slight discomfort. You will be given instruction about how to prepare the probe, and will self-insert the probe in a private

room. You will be provided with water-based lubricant if necessary, and will secure the probe with a soft gauze “sumo sling” harness which will keep it in place during exercise. There is a slight but real risk of perforation of the bowel from the insertion of the rectal probe, though the investigators are unaware of this ever occurring in a research setting. There is also a chance that surface electrodes or electrode tape may cause some skin irritation.

Because of the duration of the exercise test (90 min), you can expect to experience fatigue and some degree of sweating. You will not be able to hydrate during the test, but you will be given as much fluid as desired at the end of the experiment. If you experience any unusual symptoms after completing a testing session, you should immediately seek medical attention and inform Dr. Cheung. The investigators will also contact you the evening of your participation to ensure that you are in a healthy state. Depending on your health status, you may be asked to consult with a physician.

RECTAL PROBE

When performed in a healthcare setting, insertion of the rectal probe is a controlled act as set out in the Regulated Health Professions Act. While this act does not extend to research outside of a healthcare setting, you should be aware of the following potential risks:

- Insertion of the rectal probe can stimulate the vagus nerve which can cause slowing of the heart rate which may lead to fainting. This is more likely to happen if you have a low resting heart rate.
- Perforation of the bowel can lead to peritonitis, a serious infection of the abdominal cavity.

You should not participate in this research if you are pregnant, are under the influence of alcohol or other sedating substances (tranquilizers, sleeping pills, street drugs) or have any history of fainting or heart disease.

CONFIDENTIALITY

Access to this data will be restricted to Dr. Cheung and the two co-principal student investigators, Mr. Geoffrey Hartley and Mr. Matthew Smith. Your participation will remain confidential. The data collected from this investigation will be kept secured on the premises of the Department of Physical Education and Kinesiology (PEKN) at Brock University in Dr. Cheung’s office or laboratory, and will not be accessed by anyone other than the listed investigators. The data (paper and electronic) will be destroyed five years after the publication of the results of the study.

Investigators will require disclosure of your name and contact information (phone, email), and therefore your participation is not anonymous during the conduct of the research. All participants will have their names removed from any data. The master list matching participants to data will be kept by Dr. Cheung, and will be destroyed following the publication of data.

All information you provide is considered confidential; your name will not be included or, in any other way, associated with the data collected in the study. Furthermore, because

our interest is in the average responses of the entire group of participants, you will not be identified individually in any way in written reports of this research.

VOLUNTARY PARTICIPATION

Participation in this study is voluntary. If you wish, you may decline to answer any questions or participate in any component of the study. Further, you may decide to withdraw from this study at any time and may do so without any penalty or loss of benefits to which you are entitled. Participation, non-participation, or withdrawal from the study will not affect your standing at Brock University.

PUBLICATION OF RESULTS

Results of this study may be published in professional journals and presented at conferences, but your personal information and participation will remain confidential. Approximately one month after we finish testing all participants, we will provide you with a summary of your own results and also the overall group results. Feedback about this study will be available from Dr. Stephen Cheung (stephen.cheung@brocku.ca, 905-688-5550x5662).

CONTACT INFORMATION AND ETHICS CLEARANCE

If you have any questions about this study or require further information, please contact the Principal Investigator or the Faculty Supervisor (where applicable) using the contact information provided above. This study has been reviewed and received ethics clearance through the Research Ethics Board at Brock University (insert file #). If you have any comments or concerns about your rights as a research participant, please contact the Research Ethics Office at (905) 688-5550 Ext. 3035, reb@brocku.ca.

CONSENT FORM

I agree to participate in this study described above. I have made this decision based on the information I have read in the Information-Consent Letter. I have had the opportunity to receive any additional details I wanted about the study and understand that I may ask questions in the future. I understand that I may withdraw this consent at any time. My participation, non-participation, or withdrawal from the study will not affect my standing at Brock University.

Name: _____

Signature: _____ Date: _____

Thank you for your assistance in this project. Please keep a copy of this form for your records.

Appendix B: Borg Scale of Perceived Exertion

The ratings of perceived exertion (**RPE**) takes into account all that you are perceiving in terms of fatigue, including psychological, musculoskeletal, and environmental factors.

This level of perceived physical effort is assigned a rating from the scale below:

RPE

6	
7	very, very light
8	
9	very light
10	
11	fairly light
12	
13	somewhat hard
14	
15	hard
16	
17	very hard
18	
19	very, very hard
20	

On this scale, an RPE of 12 to 13 corresponds to approximately 60 to 79 percent of maximal heart rate. An RPE of 16 would correspond to about 90 percent of maximal heart rate. Thus, as a rule, most folks would exercise between 12 and 16 on this scale.